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OF OPHTHALMOLOGY

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 New York, N. Y.
 San Francisco, Calif.
 Scranton, Pa.
 Great Falls, Mont.

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| Cowin, Carl C., | Jamestown, N. D. |
| *Cowper, H. W., | Buffalo, N. Y. |
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| *Curtin, Thos. H., | New York, N. Y. |
| *Curtis, Elbert A., | Newark, N. J. |
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| *Davis, Frederick A., | Madison, Wis. |
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| Diem, Oscar, | New York, N. Y. |
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| Donavan, J. A., | Butte, Mont. |
| Donnell, N. R., | St. Louis, Mo. |
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| Dorsey, J. G., | Wichita, Kans. |
| Dow, Frank E., | Northampton, Mass. |
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Israel, N. E.,
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 Jacobs, M. W.,
*Jacoby, D. P. A.,
*Jameson, P. C.,
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*Jennings, Chas. W.,
*Jennings, J. Ellis,
 Jervey, J. W.,
 Jessaman, L. W.,
 Jobson, George B.,
 Johnson, Walter B.,
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*Johnston, J. G.,
 Johnston, Wilson,
 Jones, Arthur C.,
 Jones, Chas. J.,
*Jones, E. L.,
*Jones, Elgin W.,
 Jones, Fred W.,
*Jones, L. Leroy,
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*Keiper, George F.,
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*King, William Rufus,
 Kirk, Albert W.,
 Kirkendale, John,
 Kirkpatrick, S.,
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 Knipe, J. C.,
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 Koller, Carl
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- Kress, George H.,
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 LaForce, E. F.,
 Lakin, H. P.,
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 *Langdon, H. Maxwell,
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 *LeFevre, S.,
 Lefler, Anna B.,
 *Lemere, H. B.,
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 *Lent, E. J.,
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 Lidikay, Chas. J.,
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 *Loeb, Clarence,
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*Monson, S. H.,
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*Moore, G. A.,
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*Moore, T. J.,
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Morton, Howard,
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Hibbing, Minn.
Minneapolis, Minn.
Kansas City, Mo.
Fort Smith, Ark.
San Antonio, Texas
Houston, Texas
Washington, D. C.
Chicago, Ill.

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| Murray, Alfred N., | Chicago, Ill. |
| *Murray, Wm. R., | Minneapolis, Minn. |
| *Myers, Dean, | Ann Arbor, Mich. |
| Myers, Harry L., | Norfolk, Va. |
| Mytinger, Geo. S., | Chillicothe, Ohio |
| *Nabers, Samuel F., | Birmingham, Ala. |
| Nance, Willis O., | Chicago, Ill. |
| *Nardin, W. H., | Anderson, S. C. |
| *Nebinger, Reid, | Danville, Pa. |
| *Neeper, Edw. R., | Colorado Springs, Colo. |
| *Neher, Edwin M., | Salt Lake City, Utah |
| Nelson, Chas. F., | Cleveland, Ohio |
| Nelson, Louis, A. | St. Paul, Minn. |
| Newcomb, John R., | Indianapolis, Ind. |
| *Newhart, Horace, | Minneapolis, Minn. |
| *Newell, Wm. S., | Washington, D. C. |
| *Neulen, E. Nelson, | Astoria, Ore. |
| North, Nelson L., | Brooklyn, N. Y. |
| *Norton, D. C., | Manchester, N. H. |
| Noyes, Guy L., | Columbia, Mo. |
| *Nutter, C. F., | Nashua, N. H. |
| Nutting, R. J., | Oakland, Calif. |
| *Oberdorfer, Archie L., | New York, N. Y. |
| *O'Brien, John I., | Schenectady, N. Y. |
| O'Brien, Steve A., | Mason City, Ia. |
| O'Brien, T. A., | Philadelphia, Pa. |
| *O'Connor, Roderic, | San Francisco, Calif. |
| O'Donnell, Major G. A., | Fort Sill, Okla. |
| *Oertel, T. E., | Augusta, Ga. |
| *Offutt, W. N., | Lexington, Ky. |
| *Ohly, John H., | Brooklyn, N. Y. |
| *Orcutt, D. C., | Chicago, Ill. |
| *Overbay, F. A., | New Orleans, La. |
| *Owen, Arthur E., | Lansing, Mich. |
| Owen, Frank S., | Omaha, Neb. |
| *Paganelli, Terigi R., | Hoboken, N. J. |
| *Park, J. Walter, | Harrisburg, Pa. |
| *Parker, Walter R., | Detroit, Mich. |
| Pasternacki, B. W., | Detroit, Mich. |
| Patterson, E. W. E., | Grand Rapids, Mich. |
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| *Payne, S. M., | New York, N. Y. |
| Peabody, H. C., | Webster, S. D. |
| Pearson, Geo. J., | Burlington, Iowa |
| Pearson, W. W., | Des Moines, Iowa |
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| Pelle, Harry L., | Louisville, Ky. |
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| Perry, Richard W., | Seattle, Wash. |

- *Peter, Luther C.,
 *Peterman, H. F.,
 Pfingst, A. O.,
 Phelps, Kenneth A.,
 Phillips, Frank A.,
 Phillips, William H.,
 *Place, E. Clifford,
 *Pole, S. Boyce,
 Pontius, Nevin D.,
 Pontius, Paul,
 Porter, Edwards H.,
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 *Posey, William Campbell,
 Post, Lawrence T.,
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 Potter, W. W.,
 *Pratt, Fred J.,
 Presnell, C. W.,
 *Price, N. W.,
 *Prince, A. E.,
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 Prout, J. S.,
 *Pusey, Brown,
 *Pyle, Wallace,
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 *Quackenboss, Alex.,

 *Radcliffe, McCluney,
 Ralston, W. W.,
 *Rand, Prof. Gertrude,
 Randolph, Wilson,
 Ravdin, M.,
 Ray, Victor,
 Reaves, W. P.,
 *Redding, Leonard C.,
 *Reed, Charles L.,
 Reed, J. Ross,
 Reeder, J. E.,
 *Reese, Robert G.,
 Reger, Harry S.,
 Reynolds, H. G.,
 *Rhode, Homer J.,
 *Rice, John E.,
 Rideout, Wm. J.,
 Riker, John D.,
 *Rindlaub, Martin P., Jr.,
 *Ring, G. Oram,
 Ringle, C. A.,
 Ringueberg, Eugene,
 Risley, J. N.,
 Ritter, Howard M.,

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† Deceased.

- Robbins, Elmer E., Jr.,
Robertson, Edwin M.,
*Robinson, H. T.,
Robinson, J. La Rue,
*Robinson, R. E.,
*Roebuck, J. Paul,
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Rogers, T. Avery,
*Rood, L. C.,
Rosebrough, F. H.,
*Row, Geo. S.,
Rowland, John F.,
*Rowland, W. D.,
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Rudolphy, Jay Besson,
Rust, E. G.,
*St. Clair, Chas. T.,
Saliba, Michel,
Samuels, Bernard,
Samuels, Maimon,
Sanderson, Hermon H.,
*Sargent, A. Alonzo,
Sartain, Paul J.,
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Sauer, W. W.,
*Savage, Moses M.,
Sawyer, W. W.,
Scarlett, Hunter W.,
*Schaeffer, Prof. J. Parsons,
*Schipfer, L. A.,
*Schlichter, Chas. H.,
Schlindwein, G. Wm.,
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Schoch, Lester E.,
*Schoenberg, Mark J.,
*Schweinitz, George E. de,
Schwenk, Peter N. K.,
Scott, Lewis M.,
*Sears, Wm. H.,
Sedwick, Wm. A.,
Seely, A. C.,
Seelye, Walter K.,
*Seiberling, Geo. F.,
*Seibert, E. G.,
*Sener, Walter J.,
*Sernoffsky, I.,
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Superior, Wis.

- *Sherman, Elbert S.,
- Shields, Jas. M.,
- Shine, Francis W.,
- *Shoemaker, J. F.,
- Shoemaker, W. A.,
- *Shoemaker, Wm. T.,
- *Shreve, Owen M.,
- *Shultz, Louis A.,
- Shuman, G. H.,
- *Shumway, E. A.,
- *Shute, D. K.,
- *Siegel, Francis X.,
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- Simpson, W. Likely,
- Sims, W. S.,
- *Singleton, E. M.,
- *Skeel, H. Robertson,
- *Skirball, Jos. J.,
- *Slataper, Felician J.,
- *Sleight, R. D.,
- *Sliteler, C. I.,
- *Sloan, Henry L.,
- *Slocum, Geo.,
- *Small, Chas. P.,
- *Smart, Frank P.,
- Smith, Arthur E.,
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- Smith, Chas. L.,
- Smith, Dean S.,
- *Smith, Dorland,
- *Smith, E. Terry,
- *Smith, George,
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- Smith, Harry A.,
- Smith, Henry M.,
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- Smith, S. S.,
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- Sneed, Carl M.,
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- *Snyder, Walter H.,
- *Souter, W. N.,
- Spalding, Fred M.,
- Spalding, James,
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- St. Louis, Mo.
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- Cincinnati, Ohio
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Standish, Myles,
*Stark, H. H.,
*Stark, Jesse B.,
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Suker, George F.,
Sulzer, Gustavus A.,
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Sutherland, Fred B.,
Sutphin, Theron Y.,
*Swab, Chas. M.,
*Swan, C. J.,
Sweet, Robert B.,
*Sweet, William M.,
Swift, Geo. W.,

*Tarun, William,
Taylor, Joseph W.,
*Taylor, Lewis H.,
*Tanner, A. S.,
Tenney, John A.,
*Theobald, Samuel,
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*Thomas, Frances W.,
Thomas, Jerome B.,
Thompson, P. H.,
Thomson, Edgar S.,
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*Thorpe, Harvey E.,
*Tibbens, Clyde E.,
*Tibbet, Albert,
*Tilderquist, D. L.,

Memphis, Tenn.
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Battle Creek, Mich.
New Kensington, Pa.
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Atlanta, Ga.
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Salt Lake City, Utah
Cheyenne, Wyo.
Denver, Colo.
Minneapolis, Minn.
Cleveland, Ohio
Lexington, Ky.
Pittsburgh, Pa.
Boston, Mass.
Chicago, Ill.
Columbus, Ohio
Troy, N. Y.
New York, N. Y.
Newark, N. J.
Philadelphia, Pa.
Evanston, Ill.
Long Beach, Calif.
Philadelphia, Pa.
Seattle, Wash.

Baltimore, Md.
Tampa, Fla.
Wilkes-Barre, Pa.
New York, N. Y.
Boston, Mass.
Baltimore, Md.
Montgomery, Ala.
Columbus, Ohio
Palo Alto, Calif.
Boston, Mass.
New York, N. Y.
Mt. Vernon, N. Y.
Pittsburgh, Pa.
Washington, Pa.
Washington, D. C.
Duluth, Minn.

- *Tingley, Louise P.,
- *Tomassene, Raymond A.,
- Torney, S. J.,
- Townsend, C. E.,
- Trimble, Clarence S.,
- Tripp, Ira A.,
- Troncoso, M. Uribe,
- *Tuckerman, W. C.,
- *Tuckerman, W. H.,
- *Turner, Hunter H.,
- *Turner, Oliver W.,
- Tydings, Oliver,
- *Tyson, Henry H.,
- Upham, Helen F.,
- *Urner, M. H.,
- *Vail, Derrick T.,
- *VanHorn, Alfred,
- VanKirk, V. E.,
- Varick, Wm. R.,
- *Veasey, Clarence A.,
- *Verhoeff, F. H.,
- *Virden, John E.,
- Vinsonhaler, F.,
- Voigt, C. B.,
- VonColditz, G. Thomson,
- *Von der Heydt, Robert,
- *Wagner, Carl B.,
- *Waldeck, George M.,
- Walker, Arthur W.,
- Walker, C. E.,
- Walker, Herbert,
- Walter, Will,
- *Waltz, F. D.,
- Wandless, Henry W.,
- Walker, Clifford B.,
- Wanamaker, A. T.,
- Ward, G. Harold,
- Warner, Garden F.,
- *Washburn, John S.,
- *Watson, Henry D.,
- *Watson, J. A.,
- *Watson, R. S.,
- Weaver, E. W.,
- Weaver, T. W.,
- *Webster, David H.,
- *Weed, Harry M.,
- *Weeks, John E.,
- *Weeks, Webb W.,
- *Weidler, W. B.,
- Boston, Mass.
- Wheeling, W. Va.
- Bellingham, Wash.
- Akron, Ohio
- Emporia, Kans.
- Cleveland, Ohio
- New York, N. Y.
- Cleveland, Ohio
- Cleveland, Ohio
- Pittsburgh, Pa.
- Augusta, Me.
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- Asbury Park, N. J.
- Cincinnati, Ohio
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- Plainfield, N. J.
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- Manchester, N. H.
- Spokane, Wash.
- Boston, Mass.
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- Little Rock, Ark.
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- Chicago, Ill.
- Detroit, Mich.
- Riverside, Calif.
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- Evanston, Ill.
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- Minneapolis, Minn.
- Saginaw, Mich.
- Akron, Ohio
- Wichita, Kans.
- New York, N. Y.
- Buffalo, N. Y.
- New York, N. Y.
- New York, N. Y.
- New York, N. Y.

- Weih, E. P.,
- *Weill, N. J.,
- *Weimer, E. S.,
- Weiss, Louis,
- *Weisser, Edw. A.,
- *Wells, David,
- Welsh, D. E.,
- *Werts, C. M.,
- *Wescott, C. D.,
- Whaley, E. M.,
- *Wheeler, J. M.,
- *Wherry, W. P.,
- *Whisnant, A. M.,
- *Whitaker, Joel,
- White, Chas. P.,
- *White, Jas. W.,
- *White, Jos. A.,
- *Whitman, Lloyd B.,
- *Wible, Elmer E.,
- *Wiener, Alfred,
- *Weiner, Meyer,
- Wilbur, Edw. P.,
- *Wilder, W. H.,
- *Wilkinson, Oscar,
- Williams, Kent E.,
- *Wilmer, William H.,
- Wilson, Alpheus K.,
- *Wilson, Edgar A.,
- *Wilson, Harold,
- *Wilson, N. L.,
- Wilson, R. C.,
- *Winter, Geo. E.,
- Winter, John A.,
- *Wise, Ralph C.,
- Wolfe, Claude T.,
- *Wolff, Julius,
- Wolfner, Henry L.,
- Wood, Casey A.,
- Wood, Douglas,
- *Wood, Hilliard,
- Wood, J. Scott,
- Woodruff, F. E.,
- Woodruff, H. W.
- Woodruff, Thos. A.,
- *Woods, Hiram,
- Woods, R. H.,
- Woodson, J. M.,
- *Worrell, J. P.,
- Wright, C. L.,
- Wright, John R.,
- *Wright, J. W.,
- *Wright, R. H.,
- Würdemann, Harry V.,
- Clinton, Iowa
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- Pittsburgh, Pa.
- Newark, N. J.
- Pittsburgh, Pa.
- Boston, Mass.
- Grand Rapids, Mich.
- Des Moines, Iowa
- Chicago, Ill.
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Wyatt, O. W.,
*Wylie, C. B.,

Manning, Iowa
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*Yost, Walter M.,
*Young, B. F.,
Young, H. B.,
Young, Thomas H.,
Yudkin, Arthur M.,

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Knoxville, Tenn.
Burlington, Iowa
New Haven, Conn.
New Haven, Conn.

*Zehnder, A. Charles,
*Zentmayer, William,
*Ziegler, S. Lewis,
*Ziporkes, Joseph,
*Zvaifler, N.,

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Philadelphia, Pa.
Philadelphia, Pa.
New York, N. Y.
Newark, N. J.

AN INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

OPENING SESSION, APRIL 25, 1922

HALL OF THE DAUGHTERS OF THE AMERICAN REVOLUTION

THE Congress was opened by the Chairman of the Committee on Arrangements, Dr. W. H. Wilmer, of Washington, D. C. The following address of welcome was then made by Hon. Calvin D. Coolidge, Vice-President of the United States:

ADDRESS OF WELCOME

Mr. Chairman, Members and Guests of the Congress: At the request of your Chariman I have the privilege of extending to you a word of greeting and of welcome. You are meeting in the capital of the United States of America, in a city that bears the name of a great world figure, dedicated to stability, to civilization, and to liberty. You are gathered this morning in the national headquarters of a society which for the past represents patriotism, and for the present represents the determination to support and maintain civil government. There are six governments officially represented here, several universities and scientific societies, many delegates from different countries, many delegates from different portions of our own nation. You are holding this conference for mutual instruction, mutual help, for the alleviation of human suffering, educating each other, for men do not educate themselves—by exchange of ideas, by consultation, study and conference they educate each other in the arts and sciences and the humanities.

You have come here on an errand of mercy, desiring to make your contribution to the alleviation of human suffering. You have come in the interests of good health, which is of great economic value as well as ministering to human comfort. As a national asset there is nothing of more value than sound public health. If you were to strike down all of those things which make up our civilization, our organizations, our institutions of learning, our arts and our sciences, the last thing with which humanity would part would be the asset of good health. With that remaining, it would be possible to reconstruct all else; with

that gone, everything else that men hold of value would of necessity perish.

You have taken for your province the care and maintenance of the eye, one of the most wonderful and most delicate organs of the five human senses, one which makes the greatest contribution to human intelligence, for if the philosophers are right, that perfection of subject depends upon perfection of object, then the contribution that is made to the perfection of mankind through a clear and accurate eyesight is one of the greatest contributions made from any human source. The old Greeks understood the problem. In their language the perfect of "to see" is the present of "to know," for with them, as with us, to have seen means to know. "If therefore thine eye be single, thy whole body shall be full of light." I recall that during my grammar school days among the sentences in the old school-book given me to learn something of the importance and the use of language, there was this sentence, which has remained with me always—a sentence full of meaning, full of inspiration, especially to those who follow your profession—"The eye is the window of the soul."

But in addition to this you come with the broader, more extended purpose of scientific investigation. The world is turning to science as its refuge, as the source of its direction and as the means of its progress. You come as ministers to the truth, determined to follow it wherever it may lead, and it is under that principle that all the world, all nations and all men, are drawing together with a common purpose. You are uplifters of mankind, ministering to the general welfare of civilization. You follow the truth. In the words of my old preceptor, who never tired telling those who came under his direction and his instruction, "Truth and Freedom—Truth coming from distinct sources, and Freedom knowing no bounds but those which Truth has set."

That represents you, that represents civilization, that represents the standards of today and the hope—the eternal hope—of the morrow.

Dr. E. C. Ellett, Dr. Cassius D. Wescott, and Dr. Clarence A. Veasey were appointed a Committee to Nominate Permanent Officers of the Congress.

The Secretary called the roll of the official delegates in the alphabetic order of their respective governments, with the request that each delegate present his credentials.

CHINA:—Dr. T. M. Li (Peking Union Medical College):

It is indeed a great pleasure to me to be able to attend this International Congress that is being held in the capital city of this great country. I consider it a great pleasure and honor to be able to meet

so many eminent ophthalmologists of this world who have devoted their lives to the conservation of vision. I come from a country in which the science of ophthalmology is still in its infancy. It is my hope that some day we shall be able to extend to you an invitation to hold your session like this in my country. I bring you greetings from China and wish you the success which I have no doubt you will have.

CUBA:—Prof. C. E. Finlay, Havana:

I beg to extend greetings of the Republic of Cuba to her northern neighbor, the great Republic in whose beautiful capital we meet this morning, and to whom we are united by the bonds of lasting gratitude for the great moral and material support they extended to us during our war for independence; next, to the members of this Congress, meeting for the first time in fifteen years, and after the untimely interruption in 1914. I wish to express the hope that this meeting may be a contribution, a material advance in the particular branch of medicine in which we are interested.

FRANCE:—Prof. F. de Lapersonne (Académie de Médecine de Paris; Faculté de Médecine de Paris):

Au nom de l'Académie de Médecine de Paris, au nom de la Faculté de Médecine et de Monsieur le Ministre de l'Instruction Publique, j'adresse mon respectueux hommage à Monsieur le Président des Etats-Unis d'Amérique, et mon salut le plus cordial aux membres du Comité d'organisation du Congrès d'ophtalmologie.

J'apporte également les vifs regrets de beaucoup d'ophtalmologistes français, qui n'ont pu se rendre à Washington à cause de la distance et des difficultés matérielles de l'heure présente, mais qui sont de tout coeur avec vous.

Ils ont délégué auprès de vous un de leurs doyens d'âge qui, à défaut d'autre mérite, a celui d'être profondément convaincu de l'importance de la mission que vous accomplissez. Nos réunions périodiques, dont vous avez si heureusement renoué le cycle, marquent d'une pierre blanche les progrès accomplis par notre science ophtalmologique, mais elles ont aussi pour but de rapprocher des hommes dignes de se comprendre et par conséquent de s'aimer.

Salut et honneur au Congrès International d'Ophtalmologie de Washington.

GREAT BRITAIN:—Mr. Edward Treacher Collins (Royal College of Surgeons; Royal College of Physicians):

On behalf of the British ophthalmologists, and of the Royal College of Surgeons of England, and the Royal College of Physicians of Lon-

don, I wish to express our indebtedness to the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-Laryngology for having initiated this great Congress. I hope at a subsequent meeting to present an invitation that will help to promote the continuity of such assemblies as this for the promotion of our special branch of the profession and for the consolidation and advance of the friendship of all ophthalmologists.

HOLLAND:—Prof. G. F. Rochat (Professor of Ophthalmology, University of Groningen; President of the Ophthalmological Society of The Netherlands):

I have the honor to represent a country whose history has been closely connected with the earliest history of the United States, and we are very proud of the fact that a little trace of our blood runs in the veins of so many of your most prominent men. Therefore the Government of Holland has been very glad to send their representative to this Congress. We are very eager to make scientific relations between our nations still more cordial than they have ever been, and as a representative of my country and of the Ophthalmological Society of Holland I wish to bring you their greetings. We wish to thank the committee of this Congress for their kind invitation and to express the hope that this Congress will serve the purpose of promoting ophthalmology in particular, science in general, and better relations between the different nations in the future.

ITALY:—Dr. Salvatore Floria, of Washington, D. C.:

I thank you for your kind invitation to be one of this illustrious scientific body. I bring to you the greetings of the Italian Government and best wishes for the success of this conference.

PERU:—Dr. Aurelio Beraun:

I have the honor to be in this country to attend this great gathering of eminent ophthalmologists of the United States and of the countries of Europe, and it is a great honor to me.

SPAIN:—Dr. F. Poyales (Faculty of Medicine, Central University, Madrid):

España me envia à la gran nacion Americana, en representacion de todos los oculistas españoles, las oculistas Norte-Americanos han invitado a un ilustre oftalmologo Español el Dr. Barraquer para mostrar sus procedimientos operatorios, he de unir al saludo entusiasta à todos los Congresistas del Internacional de Oftalmologia

en Washington, nuestro proposito de colaboracion al progreso de la ciencia oftalmologica en favor de la humanidad.

SWEDEN:—Prof. Allvar Gullstrand, Upsala:

The Swedish Government, by sending an official delegate to this Congress, has shown how it appreciates the high level of scientific research and of medical and ophthalmological work in this country. I came here with high expectations, but I must tell you that, having been here for a couple of weeks, I have learned and seen so much, I have found the level so high and all the colleagues so friendly, that I have gotten a mighty impression of this country. I want again to congratulate the United States on having such a staff of scientific ophthalmologic men.

BELGIUM:—Prof. Emile Gallemaerts (Université de Bruxelles; Société Belge d'Ophthalmologie):

I shall take the opportunity offered by this occasion to say a few words of thanks for the welcome you have given us. Belgium had the honor of first proposing the organization of An International Congress of Ophthalmology, and I now have the honor of bringing the salutation of the Belgians to your noble and generous country. The difficulties which you had to meet in the organization of this meeting did not discourage you; the number of subscribers, the list of announced communications, prove that success has resulted from your effort. This success shows the necessity of international meetings. After being side by side for the defense of justice, honor, and humanity, we shall meet each other hand to hand in science and work in the same way for the progress of our beloved specialty. Let us hope that this meeting, which will mark a new milestone in the history of ophthalmology, will be the starting-point of a long series of meetings which will never again be interrupted.

SCOTLAND:—Dr. George Mackay (Vice-President, representing Royal College of Surgeons, Edinburgh):

I have come a long way, but I do not propose to detain you for many minutes. I gladly take the opportunity you afford me, sir, to convey to you from the most ancient medical corporation of the British Isles, the Royal College of Surgeons of Edinburgh, founded in 1505, their hearty greetings and congratulations on your great effort to revive once more these important international meetings. Our warmest sympathies go out to you, and we feel confident that this will reorganize once more those friendly and helpful meetings which meant so much for the progress of ophthalmology.

BRAZIL.—Dr. Cesario de Andrade and Dr. Pimental Franco (Faculdade de Medicina da Bahia, Sociedade Medica dos Hospitan da Bahia, Brazil).

ARGENTINE:—Dr. Francisco J. Soriano (Asociación Médica Argentina).

Lt.-Col. F. H. Garrison, Army Medical Corps, United States; Lt. James F. Hooker, Navy Medical Corps, United States; Surgeon John McMullen, Public Health Service, United States.

REPORT OF COMMITTEE ON NOMINATIONS

The Committee on Nominations submitted the following names as permanent officers of the Congress:

President:

DR. GEORGE E. DE SCHWEINITZ, Philadelphia, Pa.

Vice-Presidents:

DR. FRANCISCO J. SORIANO, Argentine.

PROF. EMILE GALLEMAERTS, Belgium.

DR. CESARIO DE ANDRADE, Brazil.

DR. PIMENTAL FRANCO, Brazil.

DR. T. M. LI, China.

PROF. C. E. FINLAY, Cuba.

DR. WILLIAM Z. HONS, Czecho-Slovakia.

MR. E. TREACHER COLLINS, England.

PROF. F. DE LAPERSONNE, France.

PROF. G. F. ROCHAT, Holland.

DR. SALVATORE FLORIA, Italy.

DR. AURELIO BERAUN, Peru.

DR. GEORGE MACKAY, Scotland.

PROF. IGNACIO BARRAQUER, Spain.

DR. D. FRANCISCO POYALES, Spain.

PROF. ALLVAR GULLSTRAND, Sweden.

DR. LUCIEN HOWE, United States.

DR. EDWARD JACKSON, United States.

DR. SANTOS DOMINICI, Venezuela.

Secretary-Treasurer:

DR. LUTHER C. PETER, Philadelphia, Pa.

Secretaries:

DR. ALBERT LASALLE, Montreal, Canada—French.

DR. FRANCISCO M. FERNANDEZ, Havana, Cuba—Spanish.

DR. JESUS M. PENICHET, Havana, Cuba—French and Spanish.

Upon motion of Dr. E. C. Ellett the report was adopted and the officers declared unanimously elected.

The President of the Congress was then introduced by the Chairman of the Committee on Arrangements.

Ladies and Gentlemen of the Congress: In high appreciation of the honor conferred, and with a keen sense of the responsibilities involved, I beg, in representative capacity and personally, to extend a hearty welcome to all here assembled; and to convey to the Chairmen and members of the Committees heartfelt thanks, in which, I am sure, all now gathered together freely join, for their insistent, unselfish and admirable work, which has been translated into effective results of which we are the happy beneficiaries. If success shall be our portion, it will be due, not to any one man or group of men, but to the fine spirit of loyalty which has pervaded all men and all women, who, striving in perfect sympathy and uncontending equity, have made possible this Congress of ophthalmologists assembled for the purpose of interchange of scientific thought, and the presentation of the results of research and of clinical observation.

For you, gentlemen, who have come from afar (I have it not in my heart to say from foreign countries, for by your very presence here you permit us to erase that word from our vocabulary)—for you confrères, colleagues and friends, I desire to sound a special note of welcome, and to reveal to you our sense of deep obligation in that you are with us. From the clinics and laboratories of the countries of the old world we of this new world have drawn a rich income—the support of our scientific existence. Our debt is large; we happily acknowledge it; we gladly owe it. We hope only that we have not been, and shall not be, either unworthy or unmindful of these benefits.

We are glad because from the Far East, from the great Province on our north, from the countries to the south of us, and from our Island neighbor, representatives have come and joined with us in this enterprise, and to them we extend our grateful welcome.

Ladies and Gentlemen of the Congress, believing that I express our united hopes, I trust we may so respect our material that it shall leave our hands finished with the master's touch; that as we proceed along the road of scientific effort our feet shall neither stumble nor hesitate; and that great success shall attend us in all our endeavors.

SCIENTIFIC PAPERS AND ADDRESSES

DES ENSEIGNEMENTS DE LA GUERRE EN CHIRURGIE OCULAIRE

PROFESSEUR F. DE LAPERSONNE

Paris, France

La Guerre mondiale, en nous appelant à soigner un nombre considérable de blessés, nous a permis de mettre à l'épreuve et de juger sous un angle nouveau les méthodes chirurgicales que nous avons employées jusqu'alors. Placés le plus souvent dans des conditions très défavorables, nous avons dû nous adapter au milieu dans lequel nous soignons nos blessés. D'autre part le contact journalier avec les Chirurgiens nous a permis de comparer leurs méthodes nouvelles et d'en faire une large application.

Je voudrais présenter au Congrès International d'Ophtalmologie quelques observations qui m'ont été suggérées par la pratique de plus de quatre ans de guerre et qui résument plusieurs travaux publiés avec nos Collègues Français. Je serais heureux si cette modeste contribution devenait le point de départ d'une discussion dans laquelle les Ophtalmologistes éminents, réunis dans ce Congrès, venaient nous apporter le fruit de leur pratique chirurgicale des blessures orbito-oculaires, et si nous pouvions ainsi établir le bilan des progrès accomplis.

Un premier fait, qui domine la chirurgie oculaire de guerre, et que nous retrouverons dans les grands traumatismes du temps de paix, c'est que les effets de ces traumatismes sont rarement localisés à l'oeil ou à l'orbite, qu'ils dépassent beaucoup notre habituel domaine et qu'ils produisent de vastes délabrements cutanés, des fractures compliquées du crâne et des cavités de la face avec corps étrangers plus ou moins volumineux.

Toutes ces blessures imposent une décision prompte et des interventions graves et délicates dont l'ophtalmologiste doit prendre la direction avec l'assistance du chirurgien et de l'otorhinologiste. Dans les services militaires que j'ai dirigés, comme dans les formations sanitaires de l'avant, j'ai toujours préconisé ces collaborations qui nous ont été précieuses.

La variété des blessures orbito-oculaires est telle qu'elles se prêtent mal à une classification. On peut cependant établir quelques catégories ayant leurs indications distinctes.

Ce sont d'abord les plaies craniofaciales avec vastes délabrements cutanés des paupières et des régions périorbitaires mettant à nu les os de la face et du crâne avec fractures esquilleuses et enfoncement. Ou bien la peau est à peine déchirée, mais on voit une tuméfaction considérable de la région orbitaire et des parties voisines, une exophtalmie très marquée avec coloration noirâtre des téguments due à de vastes hématomes, le tout souillé par du sang caillé, de la terre ou des corps étrangers des plus divers.

Derrière ces téguments profondément contus, des fractures multiples intéressent les parois orbitaires. Par ordre de fréquence on rencontre: 1. Les fractures multiples de l'os malaire, donnant une crépitation en sac de noix, avec irradiations vers l'apophyse zygomatique, le maxillaire supérieur ou l'apophyse orbitaire externe. 2. Les fractures du maxillaire supérieur avec enfoncement du sinus maxillaire. 3. Les fractures du rebord supérieur de l'orbite, véritables fractures de la base du crâne avec toutes leurs conséquences. 4. Les fractures supéro-internes intéressant les sinus frontaux et ethmoïdaux.

Les corps étrangers orbitaires plus ou moins volumineux sont, en outre des balles, des éclats d'obus ou de schrapnells, des fragments les plus variés entraînés par l'éclatement du projectile. Parmi les plus curieux que j'ai observés je citerai le cas d'une bague en cuivre de détonateur, mesurant six centimètres de diamètre, brisée en deux demi anneaux, l'un était implanté transversalement dans les deux orbites en arrière de la racine du nez, l'autre était enfoncé verticalement dans l'orbite gauche et le sinus maxillaire. On trouve également des corps étrangers beaucoup plus petits, difficiles à repérer par la radiographie ou par l'électro-aimant, dont la recherche dans l'orbite est d'autant plus délicate.

Au milieu de ces désordres, l'oeil est plus ou moins atteint. Une violente contusion sans déchirure de la coque oculaire peut produire de grosses hémorragies, des décollements de la rétine ou la luxation du cristallin. A cette catégorie de contusions semblaient appartenir ces petites hémorragies rétinienues, discrètes, localisées au pôle postérieur et à la macula, produites par des contusions, avec ou sans fracture du rebord orbitaire supéro-externe, suivant un mécanisme dont M. Lagrange a bien dégagé les lois. A peine visibles à l'oph-

talmoscope, elles ont cependant des conséquences assez graves sur la vision.

Parmi les plaies pénétrantes, tantôt il s'agit d'un véritable éclatement du globe, dont il ne reste que des débris de membranes au milieu de caillots; tantôt il y a une plaie pénétrante plus ou moins irrégulière, scléro-cornéenne ou postérieure, avec des désordres masqués par l'épanchement sanguin presque total; tantôt enfin la plaie est très petite, à peine visible.

Les corps étrangers pénétrant dans la cavité oculaire à la faveur de ces plaies sont d'un volume très variable. Mais une mention toute spéciale doit être réservée aux petits éclats intra-oculaires sur lesquels j'ai attiré l'attention avec plusieurs de mes collègues. Fréquemment observés pendant la guerre de tranchée et dans les combats à la grenade, ils pénètrent par une plaie à peine visible et vite cicatrisée. Uniques ou multiples, magnétiques ou parcelles de bois, de pierre ou de verre, leur gravité est grande. En raison de la difficulté de leur repérage par la radiographie et des épreuves négatives par l'électro-aimant, ils ne peuvent pas toujours être extraits immédiatement et ils entraînent des iridocyclites plastiques avec tout au moins perte fonctionnelle et atrophie du globe.

C'est pour cette catégorie de blessures que nous avons été obligés de faire le plus d'énucléations secondaires, afin d'éviter l'ophtalmie sympathique.

Sans plaie pénétrante, de petits éclats s'incrustent sur la surface cornéo-conjonctivale, produisant une réaction violente, même après ablation méthodique et soins antiseptiques. Peu de jours après il peut survenir une iridocyclite très douloureuse qui entraîne la perte de l'oeil. On pense à la pénétration méconnue de quelques petits éclats, ou si l'autre oeil a été atteint, on conclut à une ophtalmie sympathique. Quelques faits suivis avec soin me permettent de dire qu'il n'en est rien. C'est particulièrement dans les éclats d'obus ou de mines à courte distance, lorsque la face et les yeux sont criblés de poudre et de petits éclats, que l'on voit se produire ce phénomène. Dès il y a plus de 30 ans, étant à Lille, j'avais observé des faits analogues chez des mineurs à la suite de coup de grisou, et j'avais émis l'hypothèse que sous l'influence de la commotion violente produite par le déplacement d'air, il se faisait des hémorragies interstitielles du tractus uvéal favorisant la production de l'iridocyclite. Ce que nous savons aujourd'hui des graves altérations cellulaires

dans le choc traumatique, nous permettent de mieux expliquer la production de ces iridocyclites fatales sans plaie pénétrante.

Dans ces différents traumatismes quelles sont les complications les plus fréquentes que nous avons à craindre?

Ce que je viens de dire des irradiations des fractures vers le crâne, fait prévoir que les plus immédiatement redoutables sont les méningites, et les méningo-encéphalites traumatiques. Nous avons observé plusieurs cas dont l'évolution a été très différente. Tantôt la méningite était suraiguë avec élévation considérable de la température, délire, contractures et convulsions, bientôt suivis de coma. La mort survenait au bout de deux ou trois jours; à l'autopsie on trouvait une forte injection méningée, un aspect louche de la pie-mère à la base, mais pas de pus. Tantôt la méningo-encéphalite avait une allure plus insidieuse; après un début violent, les phénomènes paraissaient s'amender, mais il persistait de la céphalée avec raideur de la nuque et souvent un écoulement intermittent de liquide céphalo-rachidien; la courbe thermométrique présentait de grandes oscillations avec périodes de torpeur, de somnolence faisant suite à l'excitation, un amaigrissement rapide se produisait et la mort survenait au bout de trois ou quatre semaines, et même plus tard. Tantot enfin des interventions heureuses permettaient d'obtenir la guérison des blessés paraissant les plus gravement atteints mais ils conservaient pendant longtemps des troubles cérébraux, et ils se sont comportés depuis comme des trépanés.

En opposition avec les grandes complications observées dans les blessures des membres et du tronc, le tétanos céphalique a été très rare grâce à l'emploi précoce et très large du sérum préventif antitétanique. Nous ne connaissons pas de cas de gangrène gazeuse dans les plaies craniofaciales.

Les blessures orbito-oculaires sont rarement infectées primitivement par les corps étrangers, nous n'avons suivi que trois blessés chez lesquels des fragments de vêtements, d'étoffe ou de cuir, avaient été entraînés dans l'orbite; il se produisait des cellulites orbitaires avec écoulement très fétide, état général grave. Ils finirent cependant par guérir avec des cicatrices irrégulières.

L'infection de ces plaies est le plus souvent secondaire; elle est due au pneumocoque, ce qui n'est pas étonnant en raison des larges communications des plaies orbitaires avec les fosses nasales et les sinus. Elle est due également au staphylocoque et surtout au streptocoque.

Chez les nombreux blessés qui nous ont été envoyés au début de la guerre, plusieurs jours et même plusieurs semaines après le traumatisme, nous avons observé fréquemment, après la chute des escarres et dans la longue période de suppuration, des érysipèles et des lymphangites. Malgré l'apparente gravité de ces complications au début, la terminaison a été favorable, mais des récidives se sont produites, entraînant finalement des cicatrices étendues, rendant plus difficile une restauration prothétique. Contre ces complications le permanganate de potasse, l'eau oxygénée, les solutions hydrargyriques, employées tout d'abord, ne nous ont donné que de médiocres résultats, les différents sérums polyvalents ont eu des fortunes diverses, c'est en somme les hypochlorites sous forme de liquide de Dakin ou de solution de chloramine, très facilement tolérés par la conjonctive et les tissus de la face, qui nous ont donné une plus grande satisfaction.

La complication locale la plus redoutée des blessures oculaires devait être l'ophtalmie sympathique. Ce que nous savions par les statistiques des guerres antérieures, aussi bien que par les accidents de travail du temps de paix, devait éveiller de grandes craintes et nous faire penser qu'elle serait fréquente.

Il n'en fut rien, heureusement; l'iridocyclite sympathique étaient rarement signalée dans les premières années de la guerre, si bien qu'on alla jusqu'à la considérer comme négligeable et ne justifiant pas des opérations aussi mutilantes que l'énucléation.

Une statistique basée sur 39 cas, communiqués par les chefs des centres militaires ophtalmologiques et publiée par M. Morax en 1917, vint remettre les choses au point; chacun de nous d'ailleurs observait dans le même temps des ophtalmies sympathiques dues à des plaies par accidents industriels. Les principales conclusions tirées de cette statistique étaient que le caractère des blessures, la présence ou l'absence d'éclats intra-oculaires, ne paraissaient avoir aucune influence sur l'évolution de l'ophtalmie sympathique, et que pour être préventive, l'énucléation devait être pratiquée dans les quinze jours qui suivaient l'infection du globe oculaire. Toutes les opérations partielles devaient être rejetées lorsqu'il y avait à craindre ces accidents. Une observation de M. Kalt montre qu'une éviscération, pratiquée quatre jours après le traumatisme, n'avait pas empêché les accidents du côté opposé. L'ophtalmie sympathique était d'ailleurs assez souvent bénigne et dans plusieurs observations on signalait une grande amélioration par l'emploi du néoarsénobenzol.

Jusqu'à la fin de la Guerre les conclusions rassurantes de M. Morax

sont restées exactes dans leur ensemble, toutefois dans quelques cas isolés on a signalé que l'ophtalmie sympathique avait éclaté quatre jours après la blessure (Chaillous).

Si l'on compare l'énorme fréquence des blessures oculaires avec le petit nombre d'ophtalmies sympathiques constatées, on peut conclure que cette redoutable complication a été réellement très rare. Cette rareté est due en partie aux énucléations parfois très précoces et atypiques que des chirurgiens non spécialistes ont pratiquées aux ambulances du front, surtout au début de la campagne. Les soins plus judicieux donnés ensuite par des ophtalmologistes ont beaucoup contribué à éviter ce danger. Comme il y a lieu de le prévoir ce n'est pas dans les vastes éclatements du globe ou dans les panophtalmies que l'ophtalmie sympathique a été constatée, elle a été surtout observée dans les plaies pénétrantes avec petits éclats intra-oculaires.

Le traitement des blessures orbito-oculaires a bénéficié au cours de la Guerre de grandes améliorations sur lesquelles je demande la permission d'insister maintenant.

Au début la plupart des blessés arrivant trop tardivement dans les services d'ophtalmologie, nous avons dû nous contenter de lutter contre l'infection par les pansements antiseptiques, le drainage, l'extraction des corps étrangers, des fragments osseux, l'énucléation retardée. Ce traitement aboutissait à des cicatrications vicieuses et nécessitait plus tard des opérations plastiques qui obviaient plus ou moins aux mutilations de la face.

Dès que les conditions d'installation dans les hopitaux de l'avant, ou les évacuations plus rapides sur l'Intérieur l'ont rendu possible, nous avons appliqué dans la plus large mesure le traitement chirurgical précoce des plaies orbito-oculaires.

La méthode des sutures primitives, préconisée par M. Gaudier, prenait dans le traitement des grands traumatismes de guerre, une place de plus en plus importante malgré quelques oppositions. Plus que dans toute autre région elle devait avoir son application dans la chirurgie craniofaciale, en raison de la rareté des infections primitives et de la plus grande vitalité des tissus; et de fait c'est cette méthode qui nous a rendu le plus de services et a complètement transformé les résultats. Quelques publications francaises ont signalé ces faits, je citerai ma communication à l'Académie de Medecine en Décembre 1918 et l'excellent livre sur l'Ophtalmologie de guerre de mes collaborateurs MM. Duverger et Velter, paru en 1919.

Je crois devoir rappeler que le traitement précoce comprend :

I. Une exploration méthodique de la blessure permettant de se rendre compte non seulement des lésions superficielles mais surtout de l'étendue des désordres profonds, intéressant les cavités péri-orbitaires. C'est dire qu'on doit toujours avoir recours à un examen radiologique complet, radioscopique et radiographique, avec les procédés les plus exacts de repérage des corps étrangers. Elle comprend également l'emploi constant de l'électro-aimant, la nature des corps étrangers ne pouvant être prévue.

II. L'anesthésie devra être locale à de très rares exceptions près. Des instillations de cocaïne à 4% (quatre pour cent) seront réservées pour les explorations superficielles de la conjonctive. Il faut toujours avoir recours à l'anesthésie par les injections de novocaïne à quatre pour cent, en y ajoutant deux ou trois gouttes d'adrénaline au millième par centimètre cube.

Les injections seront retro-oculaires ou profondes, passant en arrière du globe et venant inonder le tissu adipeux de l'orbite dans la région du ganglion ciliaire; elles atteindront au sommet de l'orbite les nerfs nasal, frontal et lacrymal; elles iront toucher le nerf maxillaire supérieur à sa sortie du trou sous-orbitaire; des injections sous conjonctivales et de véritables barrages sous cutanés complèteront l'anesthésie totale de l'orbite et des régions voisines qui sera obtenue après dix ou quinze minutes. Je n'insiste pas sur la technique spéciale qui a été parfaitement décrite par le Prof. Duverger (de Strasbourg).

Pour nous l'anesthésie locale a presque complètement remplacé l'anesthésie générale par le chloroforme, l'éther, le chlorure d'éthyle ou le protoxyde d'azote. Depuis la Guerre, dans un service de Clinique aussi actif que celui de l'Hôtel-Dieu de Paris, c'est à peine si nous avons recours deux ou trois fois par an à l'anesthésie générale, tant pour les opérations douloureuses de la chirurgie oculo-orbitaire que pour le traitement des blessures. La quantité de novocaïne employée, même pour les anesthésies les plus difficiles, larges autoplasties ou exentérations orbitaires pour grosses tumeurs, est très inférieure aux doses indiquées pour la chirurgie générale; nous savons d'ailleurs que la toxicité de la novocaïne est faible. D'autre part il est à peine besoin de dire que cette anesthésie fait disparaître toute crainte de shock pour des sujets plus ou moins déprimés ou atteints d'affections organiques.

L'anesthésie à la novocaïne est plus difficile à obtenir lorsque les

tissus sont enflammés; il faut quelquefois attendre plus de vingt minutes avant de pouvoir intervenir et l'opération est souvent douloureuse; à plus forte raison lorsqu'il existe des fusées purulentes et des complications dont il est difficile de connaître les limites. Pour le phlegmon de l'orbite par exemple, il n'est guère possible de recourir à l'anesthésie locale. Ceci est une nouvelle raison en faveur du traitement précoce des blessures orbito-oculaires.

III. Les blessures intéressant le globe devront être abordées par la voie palpébro-conjonctivale et la conduite sera, *bien entendu, différente suivant le cas.

Je rappellerai seulement quelques principes:

1. La recherche et l'extraction des plus petits corps étrangers intra-oculaires devront être extrêmement précoces. Nous savons par les traumatismes industriels que les degrés de réussite et de conservation de la vision est en raison inverse du temps écoulé depuis la blessure. On ne peut donc indiquer aucune limite et la latitude de quarante huit heures, assignée pour certaines blessures, est ici beaucoup trop grande. La tolérance relative pour les corps étrangers est souvent trompeuse et nous avons cité plusieurs exemples de petits éclats intra-oculaires, permettant même une bonne vision, qui ont donné lieu à des iridocyclites violentes après dix huit mois ou deux ans.

Une seule exception peut être admise pour les corps étrangers intracristalliniens qu'il est préférable ou d'abandonner ou d'extraire avec la totalité de la cataracte.

Malgré tout, nous avons conservé l'impression que les corps étrangers intra-oculaires, dûs à des blessures de guerre, entraînent toujours un pronostic beaucoup plus grave que ceux provenant de traumatismes industriels. Ceci se conçoit facilement.

2. Si tout espoir de conserver tout ou partie de la fonction visuelle doit être abandonné, on devra pratiquer l'ablation du segment antérieur, l'éviscération ou même l'énucléation s'il ne reste plus que des débris oculaires. Opérant d'une façon précoce, cette dernière sera plus rarement employée, ce qui est toujours préférable au point de vue prothétique. Mais le blessé sera étroitement surveillé les jours suivants, en se souvenant que de violentes douleurs sont le meilleur signe d'une réaction inflammatoire et qu'il ne faudra pas hésiter à faire une énucléation secondaire seule préventive de l'ophtalmie sympathique.

3. Ces opérations devront être complétées par le manteau conjonctival qui pour être efficace devra être ample et bien cousu. La con-

jonctive largement libérée devra être réunie avec le plus grand soin d'un bout à l'autre au moyen de sutures séparées en U.

4. On terminera par la réfection complète et méthodique des paupières, même les plus contuses, en évitant les larges résections, en régularisant les bords des plus petits lambeaux, particulièrement au niveau de la fente palpébrale, en faisant même des autoplasties par glissement ou par renversement du lambeau, avec pedicule, si la perte de substance est trop considérable.

IV. Toutes les blessures orbitaires devront être abordées par de larges orbitotomies, même si le traumatisme a directement intéressé le globe; après avoir traité les plaies de l'oeil comme il est dit plus haut, on fera une longue incision courbe suivant le rebord osseux. Elle permettra d'aller à la recherche des corps étrangers, des fragments osseux ou des volumineux hématomes. L'orbitotomie suivant les bords inférieur et externe donne la plus large voie d'accès même sans résection de la paroi externe à la Krönlein. Mais le lieu de l'incision sera souvent commandé par les lésions osseuses. L'orbitotomie sera donc supérieure pour l'exploration de la voute orbitaire, supéro-interne pour les sinus frontaux et même franchement interne pour aborder les sinus ethmoïdaux. Elle nous a permis à plusieurs reprises de rechercher des fracas osseux et des corps étrangers situés profondément dans la partie interne de l'orbite et dans le massif ethmoïdal. Après le nettoyage soigné des parties profondes, l'incision de l'orbitotomie suturée se réunira toujours à première intention.

Telles sont les considérations qu'il m'a paru de quelque intérêt de présenter au Congrès International.

Si nous nous reportons à la chirurgie orbito-oculaire de 1914, et surtout aux méthodes employées au début de la guerre, il nous semble juste de dire qu'un progrès considérable a été réalisé. Ces résultats favorables sont dus surtout au traitement chirurgical aussi précoce que possible, aidé par l'anesthésie locale à la novocaïne, avec recherche des lésions profondes de l'orbite et sutures primitives de plaies cutanées et conjonctivales. Depuis la guerre nous avons continué à appliquer rigoureusement ces méthodes dans les traumatismes accidentels, elles nous ont donné la plus entière satisfaction.

DISCUSSION

DR. FREDERICK T. TOOKE (Montreal, Canada): It affords me pleasure, as an old student of Professor de Lapersonne, to offer him my felicitations on his beautiful presentation. It would not be fitting on my part to raise any points in discussion; but those of us who perhaps have had a little experience in war surgery, and those who have been able to follow him in his own language, notice Professor de Lapersonne's directness of thought, the same as in former years, and his beauty and simplicity of manner.

Without bringing up any point of discussion I can simply assure Professor de Lapersonne and those who have heard him that "old friends are dearest and old tunes are sweetest." Many of his observations may perhaps be well known, and the application of some of the points he has brought out, more particularly in civilian life, may be interpreted by the latter part of the quotation. I am sure I can assure Professor de Lapersonne on your behalf, as I do most heartily on my own, that from now on the first part of the quotation as to old friends will apply very directly.

DR. GEORGE S. DERBY (Boston, Mass.): I feel it a privilege to testify to the great help which we medical men of the army serving in France obtained from our French colleagues. I had the opportunity to attend the meetings of the Société Française d'Ophtalmologie, and to visit the clinics of Prof. de Lapersonne, Dr. Morax, and Prof. Lagrange, and from each place I went away with information which certainly was of great benefit to me. Among the important observations of our French colleagues were the value of thorough exploration and cleaning of wounds; the employment of sutures early as an aid to better cosmetic results; the benefits to be obtained from collaboration with those engaged in other branches of surgical work; the effects and treatment of gas cases, especially mustard gas; and the effect of contusions, either as a result of direct injury to the eye or as secondary affections—the contusions which have been so beautifully described and pictured by Prof. Lagrange.

Our experience with sympathetic disease was the same as others, and I think much was due to their teaching in regard to early exploration and treatment. It seems remarkable that the results in regard to sympathetic disease should have been so good, because in many cases the eyes were actually in pieces and it was with the greatest difficulty that the torn remnants were removed from the orbit.

The value of local anesthesia, to which Prof. de Lapersonne devotes so much attention, and the early search for foreign bodies within the eye and their immediate removal if the sight of the eye was to be saved, in any considerable number of cases, were shown in the work of his clinic. We were also impressed with the results secured by Prof. de Lapersonne and his colleagues in plastic operations, and the wonderful prosthetic appliances devised by them and which did so much to restore the good appearance of those who were mutilated in the war.

DR. ALLEN GREENWOOD (Boston, Mass.): I wish to draw attention to the fact that there probably may have been some difference between the French

soldiers and our own, particularly in regard to infection with the Bacillus Welchii. I have personal records at this time of three cases of gas gangrene of the orbit. From the three cases the Bacillus Welchii was obtained in nearly pure culture, giving a positive diagnosis of gas gangrene of the orbit. One of these men died from infection, but the other two recovered after free drainage of the orbit.

PROFESSOR G. F. ROCHAT (Groningen, Holland): C'est vraiment remarquable que sur tant de milliers de blessés de l'oeil, il se trouvent si peu de cas d'ophtalmie-sympathique. D'abord on a même cru que les cas d'ophtalmie-sympathique étaient d'une rareté exceptionnelle et il se trouvent dans la littérature française quelques communications dans lesquelles les auteurs doutent que l'ophtalmie-sympathique se soit jamais montrée. Mais, comme nous venons d'apprendre, le Docteur Morax a publié une statistique sur 39 cas. Ce qui est encore très peu. Ce résultat heureux est sans doute attribuable à l'habileté de nos confrères français et aux soins judicieux que les malades reçoivent. Mais il me semble qu'en dehors d'une signification purement pratique, ces résultats ont une importance au point de vue de la théorie, car ils parlent fortement en faveur de la théorie que l'ophtalmie-sympathique soit une infection et non pas une réaction anaphylactique due au pigment oculaire. Si l'ophtalmie-sympathique fut due au pigment oculaire, on l'aurait sûrement rencontré très souvent dans tant de milliers de blessures perforantes avec destruction de l'épithélium pigmenté. Et, d'autre part, l'observation qu'a fait M. de Lapersonne, que l'ophtalmie-sympathique s'est surtout montré dans les cas de plaie pénétrante, sans vastes éclatements, mais avec de petits corps étrangers intraoculaires, ce que s'explique facilement en admettant une infection, mais difficilement par la théorie anaphylactique.

DR. E. E. BLAAUW (Buffalo, N. Y.): Je viens justement d'arriver et n'ay pas eu le temps de me préparer. Veuillez Ms. de Lapersonne me prendre comme je suis. Permettez-moi de nous congratuler que vous nous avez donné la possibilité de renouveler la connaissance que nous avons fait au précédent congrès de Naples.

Comme marque de mon appréciation et de mon intérêt pour l'ophtalmologie française, je vous demande de me donner votre opinion sur cette question: "Quand on voit, spécialement après un traumatisme que des vaisseaux viennent invader le parenchyme de la cornée et passent aussi près de la Descemet, est-ce que vous conseillez l'énucleation immédiate?"

PROFESSOR DE LAPERSONNE (closing): Je remercie bien mes collègues, qui ont fait des observations au sujet de ma communication. Ils ont approuvé d'une façon générale, ce que nous avons observé en France. La question d'ophtalmie-sympathique n'a pas été discutée au point de vue théorique, mais il est évident qu'elle est d'origine infectieuse plutôt qu'anaphylactique. Ceci est plutôt de la théorie.

En ce qui concerne la question de l'énucleation qui doit être faite dans les blessures récentes, je crois, en effet, que toute infection ayant pour point de départ, l'iris, le corps ciliaire avec vascularisation de la cornée, est un signe très important, et il est nécessaire de faire l'énucleation le plus tôt possible.

DETACHMENT OF THE VITREOUS

SIR WILLIAM LISTER

London, England

I will begin my paper by briefly summarizing the information I have been able to gather with regard to the development and structure of the vitreous, and will then describe some of the changes which take place in it following hemorrhage or inflammatory infiltration, which are often associated with its contraction and detachment, and later proceed to my chief theme, namely, "Detachment of the Vitreous," and consider its *frequency*, its *causation*, its *effects*, and finally its *influence* on certain pathologic changes in the posterior part of the eye.

DEVELOPMENT

From the beautiful work of Mawas and Magitot it seems possible that the old view we were taught, that the vitreous was derived from the mesoblast, which entered the secondary optic vesicle through the choroidal cleft, is incorrect and that the vitreous is mainly epiblastic in origin and that its development takes place in three stages:

1. There is a primordial vitreous, formed almost entirely from the retinal epithelial cells, especially from those of the pars ciliaris, and to a very slight extent from the cells of the lens.

2. This primordial vitreous is temporarily pushed aside by the invasion of the mesoblast, both that part which comes in through the choroidal cleft and that which enters the secondary optic vesicle anteriorly with the inclusion of the lens. This mesoblast forms what they call the transitory vitreous, viz., the vascular tissue which temporarily occupies the greater part of the so-called "vitreous chamber," and ensheathes the lens, but soon disappears, remaining only as the walls of the hyaloid canal.

3. Corresponding with the disappearance of the vascular transitory vitreous, the primordial vitreous tissue undergoes great development and becomes the main vitreous body, or definitive vitreous.

Their view is, then, that the main vitreous body is epiblastic and

not mesoblastic in origin, and is derived chiefly from the retinal cells of the pars ciliaris.

In this definitive vitreous fibers are found which are continuous with and closely united to the cells of the pars ciliaris. The firm adherence of the vitreous to the pars ciliaris found in post-natal life is thus associated with its origin; while to the rest of the retina the vitreous is merely applied, or if attached, only very slightly. One would expect, therefore, that on contraction detachment would take place at the posterior region from the retina proper, with a heaping-up of the vitreous into a mass situated behind the lens and the pars ciliaris.

STRUCTURE OF VITREOUS BODY

The vitreous in its fresh state is a pellucid, soft, gelatinous mass, apparently structureless except for the so-called "corpuscles" of the vitreous, of which some show ameboid movement and contain large vacuoles. That the rest of the vitreous is not entirely homogeneous is shown by the fact that, when thrown on a filter, a small portion always remains, although by far the larger part drains away and may be collected as clear watery fluid.

When hardened and stained, a certain amount of structure can be made out. Definite fibers, already referred to, can be seen chiefly in the external part of the vitreous, and especially in the region opposite the ciliary body, to which the fibers are firmly attached. These fibers in the ciliary region are in series with the fibers of the suspensory ligament, which indeed are considered by many to be merely a highly developed group.

"HYALOID MEMBRANE."—Investing the whole of the vitreous, a "hyaloid membrane" has been described as a specialized, thin, glassy layer. Schäfer describes the hyaloid as consisting of two layers in the ciliary region—an inner one, which continues forward, the investing coat of the vitreous, and an outer fibrous layer, which is closely adherent to the pars ciliaris, and anteriorly passes forward to fuse with the capsule of the lens and forms the suspensory ligament. Of the existence of the suspensory ligament there is, of course, no question, but certain observers have doubted the existence of these other vitreous structures.

When fixed in *chromic acid*, more or less distinct lamellation of the vitreous can be seen, especially in the part next the retina, which, in the human eye, is of a firmer consistence than the central portion. In

addition to this indication of concentric lamellation a radial marking has also been observed in sections made transversely to the axis of the eyeball in similarly hardened specimens (Hanover).

Some consider that the indications of concentric lamellation and radial striation and even the hyaloid membrane are artefacts due to hardening reagents, but I think when certain pathologic states of the vitreous are examined, they afford strong supporting evidence of some such structural formation.

We may look upon hemorrhage or infiltration into the vitreous as most useful *staining reagents* of the vitreous *in situ* and *in vita*.

Now it is well known that the vitreous may undergo various degenerations; it may liquefy and lose its gelatinous character, and when liquefied, it may either remain clear—as is so often seen in cases of high myopia—or it may become impregnated with products of degeneration, such as cholesterin, as in synchysis scintillans. It would be expected that in a vitreous which had become liquefied any structural characteristics, if they existed, would be lost, and if subsequently it were infiltrated with round cells or blood, a comparatively even or general infiltration would take place; whereas if infiltration takes place in a previously healthy vitreous, some evidence of structure, if such existed, might be expected to be seen in the first stages before the whole vitreous became purulent; it is this evidence I hope to demonstrate. It is probably the anatomic structure of the vitreous which is the chief deciding factor that brings about the varied appearances of the vitreous seen on pathologic examination after effusion of blood or round cells into it.

In septic infection of the vitreous a great variety in the distribution of the round cells is seen—

(a) In some, the vitreous is evenly cloudy or evenly purulent. This may be due to the preëxisting liquefied state of the vitreous, or to the suppuration having been so prolonged that any early uneven infiltration had become lost, or to a special infection causing liquefaction of the vitreous *pari passu* with the infiltration.

(b) In others the infiltration is far from homogeneous, thus:

1. Stilling's canal may be seen demarcated in pus.
2. In other cases we see pus extending backward from the ciliary region, after anterior infection, in streaks or Medusa-like locks, while in others there is very definite and beautiful lamination of the layers of pus.

Such irregular distribution of the round-celled infiltration must be

determined by some cause, and could be determined by lamellation of the vitreous or some remains of it. Thus cases with streaks or layers of pus in the vitreous appear to give strong support to the view that the vitreous body is not originally structureless, but lamellated.

Though for years a specialized investing coat of the vitreous or hyaloid membrane has been described, others, Mawas and Magitot, for instance, state that no such structure exists.

Pathologic conditions again throw light on the matter. When looking at specimens macroscopically, with infiltration of the vitreous associated with some degree of detachment, though in some the posterior boundary of the vitreous is shaggy and there is no definite edge, in others the vitreous has a perfectly defined margin. Such definition of the vitreous must have a determining cause. It seems impossible to avoid the view that there is some specialized layer limiting the vitreous which, provided it has not been previously degenerated, is capable of checking, to some extent, the progress of invading leukocytes; but if the vitreous were degenerated and liquefied, such limiting membrane would break up and disappear, and when infiltration takes place, no definite demarcation of the vitreous is visible, but its limits are shaggy.

Another group of cases may throw some light on the structure of the vitreous, though this is doubtful, but they are interesting and afford scope for speculation.

When a foreign body has passed through the vitreous, it is not uncommon to find straight thin planes of fibrous tissue traversing the vitreous in one or more places. These planes are very surprising; doubtless they are the result of hemorrhage, but one naturally wonders why the hemorrhage should be limited to a definite plane. One would expect to find a tuft of hemorrhage rather than a defined thin sheet. I think two suggestions can be put forward in explanation:

(a) Either that the explosive force of the penetration split the vitreous just as a jelly (containing insufficient gelatin for artistic cooking) splits on a plate, or as a glacier, which we are told follows the same laws as semisolids, splits into seracs as it passes over a projecting portion of its underlying bed, or—

(b) That some radial structure of the vitreous has determined this peculiar distribution of the effused blood.

Whatever may be the exact explanation of the latter group, the examination of the pathologic specimens shown tends to confirm the

view that the vitreous has structure of a lamellated character—and also that it possesses a definite and specialized limiting membrane.

DETACHMENT OF THE VITREOUS

Detachment of the vitreous is dismissed by Fuchs in his text-book very summarily as being much less common than was thought to be the case. He writes: “. . . Such detachment is much more rare than was formerly supposed, when observers were misled by the presence of artefacts produced by the hardening of the specimens,” inferring that in most instances, when it is found on pathologic examination, it is due to the fixing reagent. That detachments are certainly not all due to fixing reagents is proved:

1. By the fact that in eyes fixed in formalin, for instance, detachment of the vitreous is not present in a large number of specimens.

In some it is present; in others, the vitreous, clear or infiltrated, may fill the whole or practically the whole of the space normally allotted to it. If the formalin caused contraction, detachment would surely be present in every case examined, except where there was some morbid attachment of the vitreous to the retina.

2. By the presence of changes, shortly to be described, which, from their complicated structure, could not possibly be postmortem artefacts, especially those found in the clear retrovitreous space unattached in any way to the vitreous body.

While it is well known that certain reagents, such as alcohol, cause *extreme* contraction, there seems no reason for thinking that formalin causes any alteration in the size of the vitreous. Detachment of the vitreous is without doubt commonly found in eyes fixed in formalin and examined pathologically, and there seems to be clear evidence that such separation of the vitreous from the retina proper has taken place during life, and is due either to actual contraction of the vitreous body or to its liquefaction, resulting, in either case, in the occurrence of a zone of nearly clear fluid between the retina and the vitreous, such as has been for long described in myopia.

In cases of the more chronic infection of the eye, all stages of contraction are met with. In the early and slighter cases, what little separation from the retina exists is only at the posterior pole, the vitreous still remaining attached to the optic disc. This is the last spot for detachment to occur in this posterior region, and it is the site at which greater adherence would be expected, since it is here that the hyaloid vessels passed from the disc into “Stilling’s canal.”

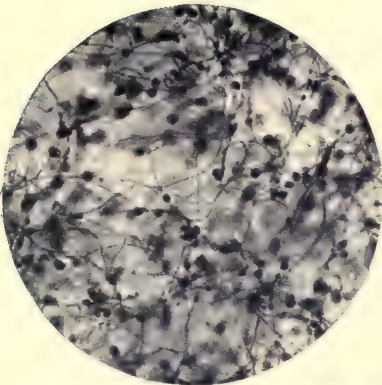


Fig. 1.—Embryonic vitreous, showing meshwork of fibers and nodes.

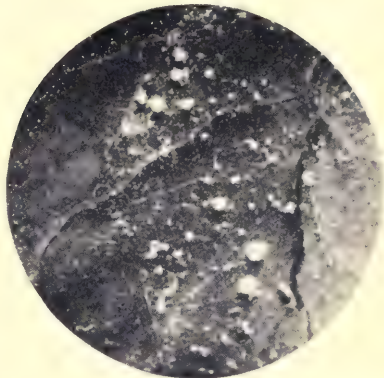


Fig. 2.—Punctate deposits on retina seen on flat, demonstrating that they have no special relation to the retinal vessels.

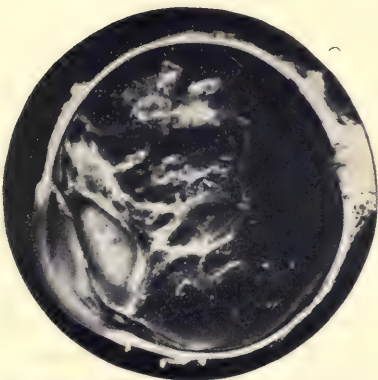


Fig. 3.—Pus extending backward from ciliary region.

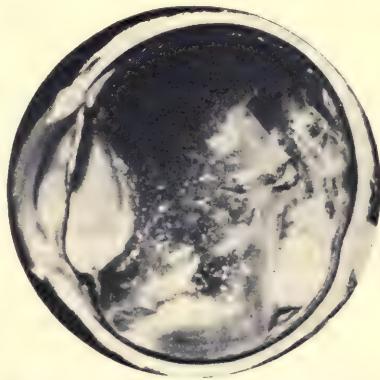


Fig. 4.—Detachment of vitreous with sharply defined margins.

In the later stages of chronic inflammation the vitreous becomes contracted up into a firm mass behind the lens.

Between these two extreme forms—the slighter and the greater—all intermediate varieties are found.

An interesting and important variety is met with when the vitreous becomes adherent to the retina at one or more spots. Such adhesion may be the result either of inflammation in the retina due to a foreign body striking it within, a perforation of the ocular coats from without, or from some other cause, such as a hemorrhage from the retina bursting into the vitreous or a patch of retinochoroiditis. When contraction of the vitreous subsequently takes place, it remains, as it were, “pegged out” at certain parts, as we shall see later.

Now, what the factor is, apart from traumatic loss of vitreous, which causes the diminution in its bulk and consequent separation or detachment from the retina to take place, it is difficult to say. We know that scar tissue tends to contract, and it is, therefore, to be expected that when fibrous tissue has replaced the hemorrhage or inflammatory exudate, contraction of such a loosely attached body as the vitreous would ensue, but is it known why scar tissue contracts, apart from consolidation and loss of fluid? It is less easy to understand why in the early stages of suppuration of the less acute types contraction is so common. Still more is it difficult to see why, when there has been no hemorrhage or infiltration, the vitreous should shrink.

I now pass on to show how detachment of the vitreous determines certain pathologic conditions.

I. SUPPURATIVE INFLAMMATIONS.—(i) When the head of the optic nerve has been irritated by toxins or organisms brought to it by the lymph-stream and pus pours out from the disc, we find quite different appearances in specimens *where the vitreous is in situ* from those *where the vitreous has been detached*. Thus in the former we have seen “Stilling’s canal” injected with infiltrate, or others where a fountain of exudate jets out into the vitreous. Whereas in similar cases of irritation of the optic disc when the vitreous has become detached a beautiful, fan-like growth of pus may extrude out from the papilla into the zone of clear fluid behind the contracted vitreous.

(ii) When the so-called “abscess of the vitreous” is formed around a retained foreign body lying somewhere in the retrolental area, the shrunk and infiltrated vitreous closely enwraps the bag of pus, while the rest of the vitreous chamber is filled with clear fluid.

(iii) In other cases, as we have already seen, when the vitreous becomes attached to the retina, we find it contracted forward but "pegged out" to the retina at certain spots.

(iv) Again, in other cases of chronic infection with detachment of the vitreous, *punctate deposits* on the retina may occur—as I described in a paper at the last Congress of the Ophthalmological Society of the United Kingdom.

II. HEMORRHAGE.—If a hemorrhage takes place into the vitreous from the retina or choroid, we know, from Parsons' work, the exact changes that occur. Either the blood elements may be completely absorbed, or if fibroblasts enter from the retina or choroid, a spindle or a fan of fibrous tissue may be formed, such as one sees in retinitis proliferans, whether of the "so-called" idiopathic or of the traumatic variety.

I have a particularly beautiful specimen of a small hemorrhage which burst into the vitreous from the retina as a result of concussion, following which the vitreous began to detach, and a pointed tag of vitreous, with a hemorrhagic tip, was left attached to the retina. As the vitreous contracted the pointed tag dragged on the retina and caused this also to detach. That the retinal detachment was present during life and not the result of the fixative is clearly demonstrated by the fact that in the post-retinal space there is an albuminous coagulum which must have been there before excision.

It seems to me one ought to lay great stress on these tags of fibrous tissue in the vitreous which are attached to the retina, for I believe they give rise to at least four separate and important phenomena. As is well known, by their traction they may cause folding of the retina, detachment of the retina, but I believe they may also give rise to holes in the retina and to the flashes which occur on movement in highly myopic eyes.

Now, it is well known that hemorrhages, especially in myopic eyes, are all too common, and, in the specimens shown, it is seen that fibrous tags may develop as a result of hemorrhage. When once they are formed, then, by their traction, not only a detachment, but I think one variety of hole in the retina, may result. There are at least five varieties of holes in the retina:

(i) The nearly circular holes which occur *at the macula* as the result of a blow, from a comparatively slowly moving, non-penetrating missile, on the front of the eye.

(ii) Other circular lacunæ *at the macula*, associated with retino-

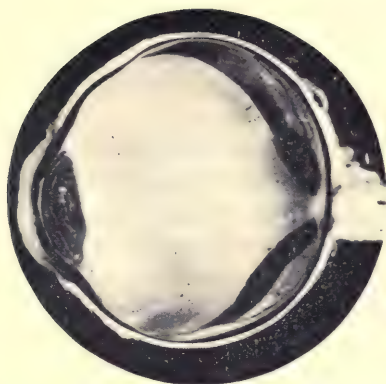


Fig. 5.—Even infiltration of vitreous with commencing detachment. Vitreous remains attached to optic disc.

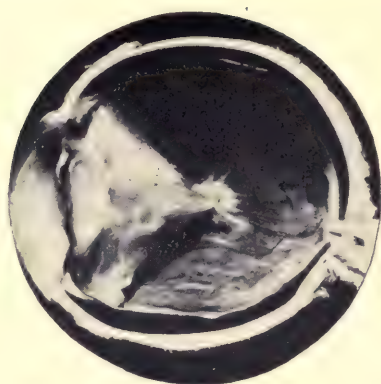


Fig. 6.—Through and through wound. Vitreous attached to retina at site of exit.

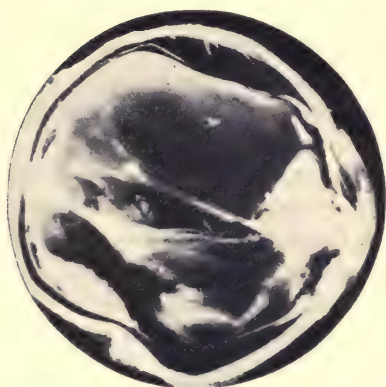


Fig. 7.—Suppuration showing Stillinger's canal infiltrated with pus and other streaks of pus in the vitreous.



Fig. 8.—Fibrous fans in the vitreous following hemorrhage, formed before detachment of the vitreous has taken place.

choroiditis, exactly resembling those following a blow, which are very probably due to bursting of cysts at the macula.

In neither of these two groups is there, as a rule, much if any detachment.

(iii) Then there are *peripheral holes* due to traumata—with or without detachment.

(iv) There are also holes associated with retinochoroiditis (elsewhere than at the macula) which has caused adhesion of the retina to the choroid. Subsequent detachment of the retina results in a rent in this membrane (Elschnig's theory). With this condition a hole may occur in two different ways—according to whether the retina is *pushed in* by fluid in the post-retinal space or *drawn in* by an adherent and contracting vitreous.

(a) When fluid collects between the retina and pigment epithelium and *pushes* the retina in, the retina may be torn at a point of attachment. Such a cause can produce only a *single hole*, for directly a breach takes place, the pressure on the two sides of the retina would be equalized.

(b) When, however, the vitreous is attached to the retina and drags it inward, then any number of holes may occur corresponding with the number of points of adhesion of the retina to the choroid.

(v) *Horseshoe-shaped Rents*.—It is these "horseshoe" rents which, I believe, are due to traction of vitreous tags, for in these we often notice that the central portion of the horseshoe is pulled inward by a tag of fibrous tissue. It would seem extremely probable that traction of such fibrous tissue attached at one end to the retina, and at the other to a contracting vitreous, would be sufficient to cause a rent.

Such a pulling in of the center of the horseshoe probably many of us have seen both clinically and pathologically.

Though it would seem possible that a steady pull might be sufficient to cause a rent, yet probably tearing could more easily be produced on sudden rotation of the eye. Thus, when a wineglass containing liquid is rotated, the glass turns before the liquid; similarly, in an eye with contracted and detached or partially liquefied vitreous, where a large part of the vitreous cavity is filled with limpid fluid, sudden rotation of the eye will cause the coats of the globe to move sooner than the fluid contents. If there is a fibrous tag attached,

on the one hand, to the retina, and, on the other, to the contracted vitreous, though *in a state of rest* it might cause little or no tension on the retina, yet *on rotation* a sudden tweak might be produced by it on the retina at the point of attachment sufficient to bring about a tear, and especially if the retina is weakened by cystic degeneration. Slighter pulls by such tags might be sufficiently forcible to stimulate the retina and cause flashes on movement of the eye.

If, then, a vitreous hemorrhage may be followed by a fibrous tag, which in time may cause detachment of the retina, we have an obvious practical indication to desist from all avoidable punctures of the vascular coats of the eye, and thus a strong reason for removing foreign bodies with the magnet by the "anterior route," through the avascular cornea, rather than by the "posterior route," through a new incision in the vascular sclerotic choroid and retina, from any of which structures a hemorrhage may pass into the vitreous.

If a hemorrhage from the disc or the retina occurs after the vitreous has become detached, quite a different appearance is found from that when the vitreous is *in situ*. Just as we have seen that pus may extend from the disc in fern-like fronds in the space behind the vitreous, so *fibrous tissue fans* may be developed in this space, which presumably are the result of a previous hemorrhage. I have an interesting specimen of a case of glaucoma with a shrunken vitreous in which a large hemorrhage from the disc was followed by a mass of fibrous tissue filling up the cup and extending into the vitreous chamber.

In grossly degenerated eyes with retinochoroiditis we find the retina glued down to the choroid, and the vitreous contracted right up behind the lens. If fibrous tissue bands occur between the retina and contracted vitreous, though the retina cannot detach, these connecting bands may be drawn out into very delicate strands. They may be either in the form of small fans or of branching filaments. Some of these are partially pigmented and occasionally contain blood-vessels.

The last section of my paper deals with what I propose to call "HOLES IN THE HYALOID."

That such a membrane or differentiation of the outer layers of the vitreous exists is, to my mind, proved, as already stated, by the fact that though in some cases of contraction and detachment of the vitreous its edge is quite shaggy and uncertain, in others it is *quite clearly defined*. Its existence is supported by other evidence, both clinical and pathologic.

1. Thus in the case of an injury from a stone a hemorrhage

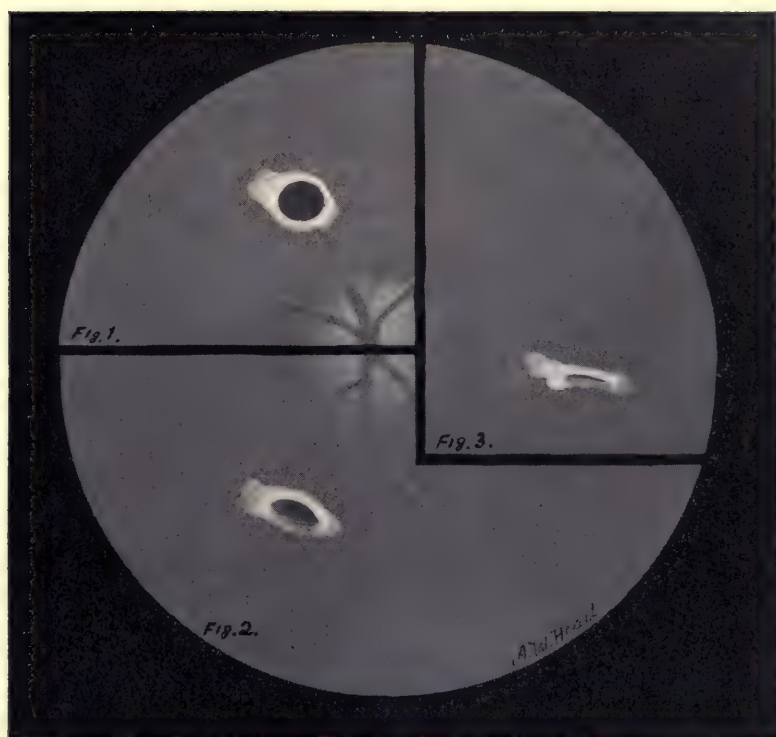


Fig. 9.—Hole in hyaloid. Opaque ring in vitreous, which on movement of eye appeared in three definite positions.

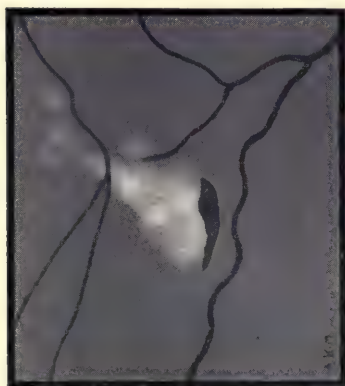


Fig. 10.—Horseshoe hole in retina. Tag of fibrous tissue attached to the intumed flap of retina, as seen with ophthalmoscope.

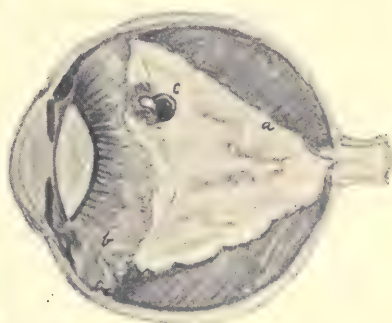


Fig. 11.—*a*, Detached retina; *b*, shrunken vitreous; *c*, hole to which is attached shrunken vitreous.

occurred at the macula which gave rise to a star figure of fibrous tissue from which radiated out multitudinous delicate rays immediately over the retina. These were much too fine to be foldings of the retina, such as one sees in cases of retinal edema, etc., and I have no doubt they were due to *foldings of the hyaloid*.

2. Again, we come across cases clinically in which, with the ophthalmoscope, a fine membrane is seen in the vitreous chamber, 3 to 6 D. in front of the retina, in which there is a clear-cut hole. I have seen three such cases and in two of them there was a patch of old retinochoroidal atrophy. The obvious explanation seems to be that the inflammatory process in the retina caused adherence of the vitreous over the patch itself and some thickening of the vitreous envelop around the patch, and when contraction of the vitreous took place, a hole was torn in the membrane around the site of adhesion, leaving a visible thickened rim to the hole.

Such holes in the vitreous might conceivably occur from tearing away of the hyaloid from either an adherent patch on the retina or from the disc itself. That the hyaloid may be torn from the disc so as to leave a hole is supported by a specimen with suppurative cyclitis which I examined pathologically, and found the vitreous contracted, and a definite round hole in its limiting envelop just opposite the disc. In the absence of any visible choroidoretinitis it is difficult to avoid the conclusion that this hole was torn from the margin of the disc, the site at which, as already noted, the vitreous is normally most adherent at the posterior pole of the eye.

The only other explanation that has been offered, so far as I know, for these cases, is that a fibrous tissue plaque has for some reason or other formed in the vitreous, as in retinitis proliferans, and that a hole has developed in it, such as is seen in those curious fenestrated layers of fibrous tissue met with lying on the surface of a retina which has been severely concussed. Against such a view I urge:

1. That in none of my cases was there any other sign of retinitis proliferans of the ordinary types, and

2. It is very difficult to imagine how a hole could develop in such a membrane lying loose in the vitreous.

To sum up my paper—the specimens which I have shown suggest or corroborate the following conclusions:

1. The vitreous has some structure and is not homogeneous, and this structure is one of concentric lamellation, as described by Hanover.

2. That there is a limiting membrane to the vitreous, *i. e.*, the hyaloid is not a myth.

3. That under certain conditions degeneration of the vitreous may occur, and both the hyaloid and all other structures of the vitreous may disappear.

4. That clinical cases are seen which can only be explained by supposing that such a hyaloid membrane has been thrown into folds.

5. That thickening in the hyaloid may take place as the result of inflammatory processes occurring in the adjacent retina, which have caused adherence of the vitreous to the retina, so that if subsequent detachment of the vitreous occurs, this limiting membrane may tear around the edge of the adherent area, and a hole in the membrane may result which is visible with the ophthalmoscope.

6. That detachment of the vitreous is common during life, and its presence in pathologic specimens is not the result of the fixing reagent, at any rate when formalin has been used.

7. That if, from any cause, the vitreous has become adherent to the retina from injury, hemorrhage, or inflammation, then, on detachment, the vitreous becomes as it were "pegged out" at certain spots, and traction is exerted on the retina which may cause flashes of light on movement of the eye, folding, detachment, or even holes in the retina.

8. That detachment of the vitreous is a necessary precursor to certain pathologic conditions, *e. g.*, punctate deposits on the retina, and the peculiar fan formations of pus or fibrous tissue which are sometimes found extending from the optic disc.

DISCUSSION

DR. M. FEINGOLD (New Orleans, La.): There is no question that the vitreous is one of the most puzzling organs in the human body. Those of us who are daily using the Gullstrand slit lamp can testify to the fact that the vitreous has some structure even in the normal eye. We see behind the lens a peculiar network, a lace-like structure, which in the normal eye has almost no movement, even with the microscope. On the other hand, the arguments brought out by the paper certainly prove that the vitreous has a structure. Hemorrhages into the vitreous and pus infiltration in a great number of cases tend to show a radial striated laminated arrangement, and I have seen some cases of injury where the radial arrangement of the hemorrhage into the vitreous was a diagnostic help, pointing to the site of the injury, showing the blood had been scattered in special directions and maintained in that position for weeks, proving that the vitreous really had some structure.

As to the question of the structure of the vitreous derived from a histologic

specimen, that is an entirely different problem, because of the point raised by the essayist of the difficulties that the eyeball presents when it comes to fixation. In a paper published some years ago from one of the German clinics the comparative value of the different fixing agents was described; there the fact was dwelt upon that the choroid in formalin fixation is greatly compressed, and the explanation given was that it was produced by swelling of the vitreous. This goes to show that we cannot at present, because of the lack of a multiplicity of fixing agents that give the same results, derive any conclusions as to the structure of the normal vitreous in vivo. As to the pathologic vitreous, there the changes are entirely different, and we can easily see with the Gullstrand apparatus distinct movement of the flakes floating in the vitreous. Whether detachment occurs in vivo is difficult to say, and we can only hope for help from this method; but that detachment is occasionally found in the histologic specimen there is no doubt. I have in my own sections seen detachment of the vitreous in which, between the detached vitreous and the retina, a fluid was found exactly as described by the essayist. I hope that we shall in the future find new fixing agents by which we will be able to study the normal vitreous, and that we shall see results from the examination of the unfixed, unhardened vitreous with the Gullstrand lamp, as has already been started by Vogt.

DR. T. B. HOLLOWAY (Philadelphia): I believe that I am voicing the sentiment of most of us present this morning when I say that we must feel very much indebted to Sir William Lister for bringing this subject to our attention. It has always impressed me that the vitreous is one of the particular portions of the eye that has been neglected—at least neglected as far as positive findings are concerned. We know so little about it. It was that fact that made me presumptuous enough to volunteer, as did Dr. Feingold, to assist in opening the discussion of this subject.

I question whether any of us today, as Sir William Lister has said, would doubt but what the vitreous has a very definite structure. We have all seen specimens that point very definitely toward a certain amount of lamination, particularly of the peripheral portion of the vitreous.

In regard to the hyaloid, I am perfectly free to confess that I had always regarded the hyaloid membrane as definitely existing until I reviewed the splendid work of Mawas and Magitot, to which the essayist has referred, and then my faith became somewhat shaken, but I have never been able to reconcile its absence with certain clinicopathologic findings.

The essayist has seen fit to refer to certain degenerative changes of the retina and in doing so has touched upon a rather tender point in my own observations. I am referring to the presence of cholesterin in the vitreous, to which he has referred. To my mind the presence of cholesterin crystals in the vitreous is an unusual manifestation of the degeneration of this structure. I think that in the majority of cases typical or atypical snowball vitreous opacities are observed.

With reference to adhesions of the vitreous to the retina and the production of holes in the retina, as I understand Sir William Lister, he thinks that a tacking down of the vitreous may occur after a focal irritation. Under these

circumstances the retina may be detached, or if a tear results, it may affect, in one instance, the retina, and in another, the hyaloid membrane, producing what he designates as a hole. Personally, I believe I have seen a tear of the first type in a case which presented itself in my service about three or four months ago, where there was a very definite lesion of this character of the horseshoe type, associated with a well-marked patch of retinochoroiditis.

He also speaks of certain foldings of the hyaloid. On the records of a case that has been under my observation for some time, I have made clinical notes to the effect that the clinical picture might well be produced by a folding of the hyaloid membrane. The lesions were central and subsequently they proved to be proliferative. However, I can readily believe that such lesions may occur.

PROFESSOR EMILE GALLEMAERTS (Brussels, Belgium): J'ai demandé la parole pour insister sur le point soulevé par le Docteur Feingold. Il n'est plus nécessaire maintenant de faire l'examen microscopique d'yeux enucléés pour étudier la structure du corps vitré à l'état normal ou pathologique. Si l'on examine l'œil vivant à l'aide de la slit lamp de Gullstrand, on voit sur un fond noir, se détacher les fibrilles du corps vitré; l'aspect de ces fibrilles varie, elles s'entr'croisent et démontrent la structure réticulaire du corps vitré. À l'état pathologique on peut observer les altérations si bien décrites microscopiquement par Treacher Collins; on voit les fibrilles changer d'aspect, s'épaissir, se raccourcir, se condenser. Il y a là un chapitre nouveau pour l'étude du corps vitré.

DR. JOHN E. WEEKS (New York City): Much light may be thrown on the structure of the vitreous body by the careful study of embryonal tissue. The embryonal vascular system of the vitreous body and the vascular system of the retina develop from mesoblastic tissue and they both develop from the same process of mesoblast that enters the embryonal retinal fissure. In the study of the embryo of the rabbit we will find that the outer surface of the vitreous is shaggy, due to minute processes that extend from it into the nerve-fiber layer of the retina, as a result of the continuity of mesoblastic tissue. The distinctly shaggy condition of the outer surface of the vitreous body in the embryo is not present beyond the ora serrata, the anterior surface beyond this point being apparently limited by the network of capillaries of which the posterior part of the membrana vascularis lentis forms the greater part. This part of the vitreous body at this stage rests against tissue that is of epiblastic origin and is not incorporated with it. In the early stages of development of the vitreous body blood-vessels permeate all parts of it. These disappear as development progresses, apparently having a very delicate framework. The capillaries are most numerous at the periphery of the vitreous body, particularly anteriorly, and here more than anywhere else the vitreous body presents a retaining structure which is apparently composed of any incomplete laminae, more closely associated near the surface of the vitreous body. This laminated membrane in the vitreous body is in effect a limiting membrane. It is not a membrane that can be seen with the microscope, as we see the limiting membrane of the lens. If we study the pathologic condition of the vitreous body,

we find that hemorrhages extending into the vitreous do so without regard to a uniform structure, and that the formation of fibrous bands do not follow any plan of structure of the vitreous. However, in certain pathologic conditions an infiltration may follow lines that indicate an irregular structure.

The holes in the so-called limiting membrane of the vitreous, I think, are only the result of pathologic material in certain areas.

DR. J. W. NORDENSON (Stockholm, Sweden): I think we are much indebted to Sir William Lister for calling our attention to the question of the detachment of the vitreous, and I only want to point out some details in his work where I think there might be some difference of opinion. There is, as you have heard, such a difference of opinion between him and the histologists who have worked on the structure of the vitreous. I wish, in this respect, to refer to the work published about thirty years ago by Gustav Retzius¹ who has shown that in his opinion the vitreous has no real membranes, but that it has a laminated structure. The laminae consist of densifications of its fibrillar tissue; and they are not concentric, but, as he has shown, are placed in a sagittal direction. These laminae, pointed out by him thirty years ago, have also been found by the investigators working with the Gullstrand slit lamp. If you take these laminae into consideration you will find an explanation not only of the clinical facts that Sir William Lister has described here, but they also seem to give a clue to the difference of opinion between him and the French investigators, Mawas and Magitot, who have worked with the vitreous. I am wondering whether what he calls a detachment of the vitreous might not be considered as a cleft or crevasse of the vitreous tissue, and what he calls the detached hyaloidea a densification of this tissue lying not in a sagittal but in a frontal plane; also if the holes that he has observed are not places where this densification is absent. I think the whole question is a little difficult to grasp if you speak about detachment of the vitreous. Might we not speak of clefts of the vitreous? Then I think the question of the hyaloid membrane would not cause any difference of opinion between histologists and clinicians.

COLONEL R. H. ELLIOT (London, England): My interest in the hyaloid was excited when I studied 750 consecutive cases of couching of the lens made by natives of India. Of these, I submitted 125 globes to anatomic examination, and in quite a considerable number a careful histologic examination was made as well. In all these operations there was, first of all, often a perforating wound of the vitreous by the instrument used in couching; second, there was disturbance of the vitreous as a result of the lens being pushed bodily into it, and third, there was septic infection of the vitreous in a large number of cases. I want you to notice one point especially, viz., that in the specimens of my series (which are lodged in the museum of the Royal College of Surgeons of England) the infection is often localized, sometimes by spread along definite planes, sometimes by strict localization to a definite area of the vitreous. Now it seems incredible that this could happen in any but an organized structure.

There is not time to take Sir William Lister's points one by one, but there

¹ Biologische Untersuchungen, No. VI, Stockholm, 1896.

are two or three I would especially like to give my support to. First, in a very definite manner my specimens indicated that the vitreous has a definite structure. Then, again, I would like to say a word, not only from my examination of the eyes I have mentioned, but also from a clinical point of view, of the existence of a definite anterior hyaloid membrane. You may speak of it as a membrane, or as a thickening of the anterior layers, but there is a definite hyaloid membrane, and that hyaloid membrane is a very definite protection (both mechanical and anti-infective) to the surgeon in a large number of operations, and especially in those for cataract.

The question of the existence of clinical detachment of the vitreous is my next point. I sectioned a number of these couched eyes shortly after their removal, having first frozen them in ice and salt. Detachment of the vitreous, as Sir William Lister has detailed it to you, was common in those specimens. On that point I am emphatic.

I speak of these matters particularly from the point of view of the clinician. It is important that we should have a clear view of the structure of the vitreous, and that we should recognize it as an organized structure with a definite limiting membrane. If we are not clear on this subject, our views will have a misleading tendency in our surgery.

PROFESSOR IGNACIO BARRAQUER (Barcelona, Spain): In my opinion the vitreous is a semisolid body, surrounded by a membrane or thickening of this structure, resistant enough to keep it isolated from the eye, and to pass it from hand to hand without losing its spheroidal form, but it can be liquefied by compressing it. Its structure is fibrillar, and with Gullstrand's lamp and the binocular microscope of Zeiss its fibers can be seen clearly contracted after an injury. The cicatricial retraction of those fibers is the cause of the detachment of the vitreous and sometimes of the retina. All detachments of the retina after cataract extraction have this origin. It is indispensable, therefore, to avoid compression of the eye during cataract operations, as well as the introduction of pointed instruments into its chambers. By doing this we shall see the disappearance of post-operative retinal detachments consecutive to wounds of the vitreous. One of the most dangerous operations is the capsulotomy, and also discission of the secondary cataract, as by them the vitreous is nearly always wounded.

DR. F. PARK LEWIS (Buffalo, N. Y.): There are two points I would like to add in supplement to Dr. Lister's observations. The manner in which the vitreous has been examined heretofore is the same as that which has been applied to other ocular structures. It has been placed in hardening fluid and sectioned and observations made. The vitreous is 90 per cent. fluid. When its contents are abstracted, the skeleton only remains and the relationships of its parts are wholly changed by compression, giving a wholly inaccurate impression of its anatomy. Certain facts, however, can be deduced from an examination even macroscopically of the vitreous when it is removed from the eyeball. The entire lenticular system comes out en masse. By placing this, consisting of the vitreous body and the crystalline lens, in slightly acidulated water it imbibes the fluid and its separate parts are more clearly brought into view. It will then be found that the vitreous is composed

of an outer exceedingly elastic membrane which balloons out to nearly one-third its normal size. But even when hardened and sectioned and placed under the microscope it will be found that the skeletonized remnant is composed of a number of fibers which curl upon themselves like shavings, showing that the internal structure of the vitreous is composed of elastic tissues. These two facts should be noted, as essential conclusions may be drawn from them.

One other point must be mentioned. I have said that our methods of examining a fluid body by first extracting the fluid in it is essentially incorrect. A method which I have found of great value is to coagulate this body without extracting its substance. By placing it in a water-box with an optically perfect glass cover its structure may be definitely determined by the aid of the Gullstrand light and the corneal microscope. Such specimens I have here in demonstration, and I will also show a photograph in which can be definitely shown some of the tubules through which the nutrition of the vitreous is maintained.

DR. F. H. VERHOEFF (Boston, Mass.): I have frequently observed separation of the vitreous on examination of pathologic specimens, and can confirm all the observations of the essayist except the finding of holes in the hyaloid. I have no doubt of their existence, but I have never observed them. I have not made a systematic study of the subject and, therefore, am not prepared to express an opinion as to its exact importance.

One of the important things about the vitreous which we do not know is exactly how liquefaction may come about. If we could produce liquefaction of the vitreous at will, we would be doing an important thing in certain cases where we have vitreous opacities. I have tried, but have not succeeded. Separation of the hyaloid membrane may play a part in certain cases of secondary glaucoma. In case of a very small sarcoma of the choroid I found the hyaloid membrane separated from the retina, allowing the vitreous and lens to be pushed forward, causing obliteration of the anterior chamber.

After any method of fixation if you stain the vitreous deeply the hyaloid membrane is seen with sufficient distinctness, it seems to me, as to leave no doubt of its existence. If you fix the retina in Zenker's fluid and then stain it in Mallory's connective-tissue stain, the hyaloid membrane will show up as definitely as the lens capsule. It does not send projections into the retina, but is entirely distinct, and can easily be pulled off from the latter. One reason perhaps that there is this difference of opinion as to the existence of the hyaloid is that the vitreous does send very fine fibers into it, but that is no reason for not regarding it as membrane. The anterior part of the hyaloid membrane, that is, the part that leaves the retina and extends behind the lens, possibly may play a part in the causation of primary glaucoma. Thus, if its permeability should become altered so there was an increased osmotic pressure behind it, the anterior chamber would be shallowed.

MR. E. TREACHER COLLINS (closing): I do not wish to take upon myself to reply in behalf of Sir William Lister, but I would like to make a few remarks myself.

I quite agree with Dr. Weeks that it is much easier to examine the structure

of the vitreous in the fetal eye than in the fully developed eye. A good many years ago I examined fetal eyes hardened in Müller's fluid and I came to the conclusion not only that the hyaloid membrane is as definite a structure as the capsule of the lens, but that the membrane in the fetal eye has cells in it scattered widely at definite distances. I think I will be able to demonstrate that to you with lantern slides made from my specimens.

Further, the vitreous of the fetal eye is permeated throughout by a network of fibers with a lot of little fine, nodular points where they cross that take on the nuclear stain. We do not know what they are; they are smaller than any known nucleus of a cell. These nuclear staining bodies disappear in the adult, but I think sometimes a few persist and float about in the vitreous, and that the *muscæ volitantes* are just remnants of this network of fibers. I have had sections of the fetal vitreous under the microscope, and at the same time have had float into my field of vision my own *muscæ*, and have thus been able to compare the appearance of floating vitreous opacities with the network in the fetal eye.

One of the most interesting points in Sir William Lister's paper is, I think, what he describes as "holes" in the hyaloid. In one of the cases in my own clinic the appearance seen seemed to me to be unexplainable on any other ground. One of these "holes" was somewhat anterior to the retina and moved about in movements of the eye. I took it that the place where the vitreous was normally attached to the optic disc had become detached and showed as a round hole.

ON DIAPHRAGM LAMPS IN OPHTHALMOLOGY

PROF. ALLVAR GULLSTRAND

Upsala, Sweden

The examination of the living eye for diagnostic purposes as well as for scientific investigations is highly facilitated by a suitable illumination of the field, and the best results are obtained when the distribution of light and shadow can be perfectly controlled. To obtain this effect it is necessary for a sharp optical image of a bright and uniform source of light to be projected on the most favorable place and other light to be shut out from the field. The latter condition requires not only a dark room, but also, in certain methods of examination, the shutting out of light that is reflected regularly by the refracting surfaces of the eye or diffusely by the substance of the cornea and of the lens.

Thus there are two conditions: the source of light must be uniform and sharply limited, with a high specific intensity, and a sharp optical image of this source of light must be procured. As is now well known,

the *slit lamp* and the *aspheric lens* (an aplanatic lens with a non-spheric surface of revolution) answer these conditions. But the slit lamp is not the only suitable lamp. What is wanted is a uniform source of light of high specific intensity adequately combined with a diaphragm through an appropriate optical system. The slit lamp is, therefore, only a special type of the *diaphragm lamp*. When the diaphragm has a circular aperture, another type results for which I suggest the name *hole lamp*.

SLIT LAMPS

That the first diaphragm lamp for ophthalmologic use was a slit lamp is due to practical reasons. Of the then available light sources, only the crater of the arc lamp and the glowing filament of the *Nernst lamp* had a sufficient luminosity combined with a sufficient area of uniformity. As the latter represented the simpler and more easily available means and proved practicable, I preferred it. As a rule, if the greatest effect is desirable, the shape of the diaphragm is determined by the shape of the source of light, and therefore with the Nernst lamp the diaphragm must be a slit. The Nernst slit lamp consists mainly of a tube which contains in one end the Nernst lamp, and which is closed at the other end by a plate carrying a slit of variable width. By means of a lens or a system of lenses an optical image of the glowing filament is procured which, by appropriate mechanical appliances, can be brought to coincide with the slit. At first I used this lamp for ophthalmometric investigations, and I have described it in the third edition of Helmholtz's handbook of physiologic optics. Then, on my suggestion, the aspheric lens was calculated by M. v. Rohr and brought on the market by Carl Zeiss in Jena. Using this lens for focal illumination with the slit lamp I immediately verified the superiority of the method, as I could see, for example, the structure of the normal vitreous body.

During the war the fabrication of the Nernst lamp ceased and good Nernst filaments cannot be bought now. Therefore in the instruments of Zeiss the Nernst lamp has been replaced by a *nitra lamp*, which consists of a glowing narrow spiral of tungsten filament in a glass bulb filled with an inert gas. This lamp has several disadvantages as compared with the Nernst lamp: the spiral is not a uniform source of light, and it is not always straight; the refraction in the glass of the bulb deteriorates the optical image projected in the slit; and the lamp demands a strong current with a low voltage,

so that an encumbering resistance must be used in the circuit in connection with an ordinary supply of electricity. For certain methods, *e. g.*, those of reflexless ophthalmoscopy, it is not essential that the source of light is uniform and the slit filled out by its optical image, so that the lamp can directly replace the Nernst lamp, but for focal illumination an optical image of a uniformly bright slit is of so high value that another device is preferable. If the lens is connected with the lamp by an arm (a device of Henker), a narrow beam of light is sufficient and in this case it is not necessary that the optical image of the spiral coincides with the slit. It can be projected on the lens by pushing the lamp deeper in the tube, whereby the optical image of the slit becomes uniformly bright.

For certain methods of reflexless ophthalmoscopy a small *incandescent lamp* is a sufficient source of light. The filament must be straight, and an optical image of it is projected in a slit in the same way as with the Nernst lamp.

HOLE LAMPS

Nowadays there is another lamp with sufficient specific intensity on the market, namely, the *pointolite lamp*, which is a tungsten arc lamp, inclosed in a glass bulb containing an inert gas at low pressure. There are several types, but the smallest of them is sufficient for ophthalmologic purpose. The lamp is lighted by a strong ionization current of short duration, but demands only a current of less than 0.5 ampere when burning. Moreover, the voltage is higher than that of the nitra lamp, and as a consequence the necessary resistance is less encumbering. But the greatest advantage over the nitra lamp is the perfect uniformity of the light source, which is a ball of tungsten used as an anode. If an optical image of this ball is projected in a circular aperture, an ideal hole lamp can be obtained. The glass bulb is sealed up on the top, and when the lamp has been used some time, deposits from volatilized tungsten are seen here on the inside. It is consequently preferable to mount the lamp perpendicularly to the optical axis of the tube. In this position the optical image of the ball is not appreciably deteriorated by the refraction in the glass bulb, and the light is less weakened by the deposits. But for many purposes the lamp can also be mounted along the axis in the same way as the Nernst lamp. It can, in these cases, replace the Nernst lamp in the ordinary slit lamp. Though the slit is then not filled out by the optical image of the ball, and though only a part of this image is

used, this combination answers well for many methods. Finally, with a special arrangement, the lamp can be used in a slit lamp for focal illumination when its optical image is projected on the lens.

FOCAL ILLUMINATION

The superiority of diaphragm lamps for focal illumination is so great that other lamps should never be used for this method of examination. In every clinical case it can be expected that something more can be seen by using the aspheric lens in connection with these lamps than with ordinary lamps and lenses. For current clinical use it is not desirable to lose time by immobilizing the head of the patient on a support for the chin. If this is not done, a rather wide cone of light is necessary to allow for the motions of the patient's head. For this reason the optical image of the source of light has to be projected in the diaphragm, as in the original Nernst slit lamp, and the uniformity of the source of light is a great advantage.

The focal illumination with diaphragm lamp and aspheric lens demands a certain amount of skill, but is soon learned with a little practice. The lens is to be held with its most curved surface turned to the light at a distance of about 40 cm. from it, and perpendicularly to the ray that passes through its center, so that the diaphragm is situated on the optical axis of the lens. That this is really the case can be controlled by projecting an optical image of the diaphragm on the cornea. This position of the lens once found, the only difficulty consists in moving the lens parallel to itself, as the optical image is projected deeper in the eye.

However insignificant this difficulty is, the device of Henker to fix the lens on an arm in connection with the lamp has probably contributed to render the method more accessible to oculists. With this device the immobilization of the patient's head by a support for the chin is desirable, but this complication offers an advantageous compensation in the possibility of using the microscope for examination. As a narrow beam of light is sufficient, the optical image of the source of light can, in the way already described, be projected on the lens, whereby the nitra lamp renders the optical image of the slit perfectly uniform.

When the pointolite lamp is substituted for the nitra lamp in this apparatus, the optical image of the slit is not uniformly bright, the middle seeming darker than the ends. This inconvenience is caused by the spheric aberration of the lens system contained in the lamp

tube in connection with the smallness of the glowing ball, and can be avoided by a special construction in which the distance between the lens system and the slit is reduced to a minimum.

The slit lamp, with the device of Henker and the Nernst lamp, the nitra lamp, or, for certain investigations, an arc lamp as source of light, has enabled Koeppe and Vogt to create a whole new branch of ophthalmology, the *microscopy of the living eye*. This is not the place to enter on the details of the results already obtained or to be expected by further investigations, but the modifications of the illuminating system ought to be mentioned. Vogt has insisted on the use of a very narrow beam of light for facilitating the localization in the third dimension. But as the slit grows smaller the chromatic aberration of the aspheric lens grows more disturbing. For this reason an achromatic combination of lenses was preferred to the aspheric lens. Such a substitution is possible because a small aperture is sufficient and, indeed, in some cases desirable for microscopic examination. Koeppe has completed the device of Henker with a tube which shuts off the light reflected in the interior of the lamp tube, with certain light filters, and with a mirror which facilitates the examination by giving the light that forms the optical image of the slit another direction than that of the optical axis of the instrument.

Moreover, for the microscopic examination of the angle of the anterior chamber Koeppe has devised both a hydrodiascopic chamber and a suitable contact lens. Another contact lens, which makes the eye highly hypermetropic, has enabled him to use the microscope for the examination of the deeper parts of the vitreous and of a certain area of the retina. Finally, with a third contact glass of annular shape and containing a conical reflecting surface, he obtains the necessary illumination for ultramicroscopy of the cornea with a dark field. He also uses polarized light for the microscopic examination of the living eye.

SIMPLE CENTRIC OPHTHALMOSCOPY

Under this name I have described a method of ophthalmoscopy of great value for current clinical work, and for which the hole lamp can be used as well as the slit lamp. In ordinary ophthalmoscopy by the direct method the corneal reflex makes it impossible to see the macula and neighboring parts of the fundus in the direction of the axis of the eye. Even though the pupil be dilated, a certain decentration of the hole of the mirror in respect to this axis is necessary to avoid the dis-

turbing corneal reflex, when this part of the fundus is examined, and this decentration is sufficient to deteriorate the optical image of the fundus, the refraction of the light being less regular in the excentric parts of the surfaces of the eye. But the diaphragm lamp affords a means of suppressing the corneal reflex and enabling the ophthalmoscopic examination of the central parts of the fundus without any decentration of the hole of the mirror in respect to the axis of the examined eye. In the light reflected by the mirror the hole acts as an opaque body and throws a shadow. If the source of light is sufficiently small, a full shadow of considerable extension can be obtained, and to suppress the corneal reflex it is sufficient that the center of curvature of the cornea (more exactly, its evolute) is situated within this full shadow. Thus, if a diaphragm lamp is used as source of light for ophthalmoscopy by the direct method, the corneal reflex can be extinguished by an adequate movement of the mirror. The observer sees a bright spot on the fundus and the corneal reflex. If care is taken that his own nose does not throw any shadow on the mirror, he needs only move the mirror so that the spot on the fundus is centered on the corneal reflex to see the latter vanish. Looking through the central parts of the refracting surfaces of the eye he then sees more details in the macula and in its neighborhood than he can see with the ordinary method. He can also perform the examination without dilatation of the pupil in cases where nothing is seen of the macula with the ordinary method. To obtain a field of reasonable extension the mirror should be concave, with a rather short radius of curvature and a narrow hole. The small mirror of Morton's ophthalmoscope answers well.

This method again demands a certain amount of skill which, however, is easily acquired by a little practice. Its advantages induce me to give here some hints. An ophthalmoscopist who is able to give the mirror the correct position after he has approached the patient till the two fronts are in contact will meet with no difficulties if he tries the method the first time on a case with dilated pupil. But many oculists are accustomed not to approach the patient until they have seen the pupil red by throwing light in it at a distance of some 15 or 20 cm. If this is the case, the lamp must be placed so far off that the cone of light extends from the patient's eye to the point from which the examiner desires to see the pupil red. If the pupil is dilated, the extinguishing of the corneal reflex by appropriate movements of the mirror is soon learned. The illuminated part of the fundus shows

a darker spot, which is situated in its center if no shadow from the nose of the observer is thrown upon the mirror, and as soon as this darker spot is centered on the corneal reflex, the latter vanishes. The spot is a half shadow from the hole of the mirror and can be avoided by bringing the lamp to a distance of 12 to 15 cm. and by using an angle of incidence of about 45 degrees on the mirror. In this way the method gives the best results, but also offers greater difficulties. The short distance diminishes the width of the cone of light at the place of the mirror, so that the observer must learn to find the correct position of the ophthalmoscope after having approached the patient; and the great angle of incidence causes a shadow of his nose on the mirror and a consecutive diminution of the illuminated part of the fundus, if special care is not taken. As in ordinary ophthalmoscopic work a smaller angle of incidence is used, it may be necessary to change the habitual position of the ophthalmoscope in respect to one's own face. The new position is easily found out by using the large angle of incidence and a greater distance of the lamp. The shadow from one's nose on the mirror is then seen on the form of the illuminated part of the fundus.

I have entered upon these details because the superiority of the method is so evident that nobody who has learned it will voluntarily use other lamps for ophthalmoscopy by the direct method. The slit lamp affords a somewhat greater field than the hole lamp.

DIAPHRAGM LAMPS IN OTHER METHODS OF EXAMINATION WITH A MIRROR

For the *examination of the transparent media of the eye by transillumination* diaphragm lamps are unrivaled. Fine dust-like opacities of the vitreous afford a striking example. It is well known that by the ordinary method such opacities can be seen with the plane mirror in cases where nothing is seen if a concave mirror is used. The usual explanation is that the light reflected by the concave mirror is too strong, but the real cause is that the plane mirror illuminates a smaller area of the fundus. Since this illuminated area serves as a source of light, and as shadows are the more conspicuous the smaller the source of light used, it is obvious *a priori* that the diaphragm lamp must give the best results. It is easy indeed to prove the sensibility of the method. If the lamp is placed at a distance of about 40 cm. and a plane perforated mirror with a narrow hole and with a convex glass of 10 or 12 diopters behind it is used, it is often possible to see

shadows which are caused by the distribution of the fluid moistening the cornea, and which change when the latter is wiped by the eyelid. Accordingly, opacities of the vitreous that, by the ordinary method, are seen only with the utmost difficulty as finest dust, appear as a sharply defined network.

In *skiascopy* the most reliable results are obtained when a transparent unperforated mirror is used. A hole in the mirror always causes a shadow which complicates the phenomena in the neighborhood of the point of reversal, and which makes it impossible to find the refraction in the central part of the pupil. Therefore a plate of glass with plane parallel faces is the best mirror, and the loss of reflected light is supplied by the specific intensity of the diaphragm lamp. This method can be regarded as an evolution of the method of Jackson or that of Wolff accordingly as the hole lamp or the slit lamp is used.

If a hole of small diameter is used as diaphragm and the mirror consists of a thin glass plate fixed in a position which allows the optical image of the hole to coincide with the observer's pupil, the sensibility of the method is highly increased and allows the examination of the aberration of the eye and of pathologic asymmetry. I have described this method under the name of *objective stigmatoscopy* in Helmholtz's handbook. With a diameter of only $\frac{1}{2}$ mm. the dilatation of the pupil can be avoided in many cases. A diameter of 1 mm. gives excellent results if the pupil is dilated.

TRANSILLUMINATION OF THE BULBUS

My successor in the chair of ophthalmology in Upsala, Professor Lindahl, has described a new method of transillumination of the bulbus with a Nernst hole lamp. He substituted a hole on the apex of a cone for the slit in a Nernst slit lamp, and used this lamp at first in the ordinary way, bringing the border of the hole in contact with the scleral conjunctiva in order to see whether the red light in the pupil was weakened by a tumor. But he found that the intensity of the light allows another method. If the cone of light is directed in the pupil, the illuminated part of the fundus constitutes a source of light sufficiently intense to show directly the shadow of a tumor and, moreover, the shadow of the ciliary body on the sclera, which appears brilliantly red. Thus Lindahl's method is an inversion of the old method for diagnosing tumors. As might be expected from the shape of the diaphragm, the pointolite lamp has proved superior to the

Nernst lamp in Lindahl's instrument. A rather large area of the fundus being illuminated, the source of light for the transillumination is large, and shadows on the sclera can be expected only from opaque bodies situated near this membrane. But the ora serrata is distinctly seen and, as a rule, the ciliary processes throw a visible shadow.

CENTRIC REFLEXLESS OPHTHALMOSCOPY

Before the construction of the slit lamp the reflexless ophthalmoscopy, developed principally by Thorner, was unable to give the best possible optical image of the fundus, inasmuch as the light that entered the eye of the observer had to be refracted in an excentric part of the optical system of the examined eye. For the same reason the maximum effect of stereoscopy could not be obtained. If the light that enters the observer's eye has to pass through the central part of the pupil of the examined eye, it can be shown that the light which illuminates the fundus must pass through a peripheral zone which, to avoid the light reflected diffusely in cornea and lens, must be the narrower, the greater the ophthalmoscopic field that is desired. This involves the use of a source of light with high specific intensity and necessitates the projection of an optical image of it in the plane of the pupil. It is not necessary that this optical image be small, as the superfluous part of it can be projected on the iris, but the optical image afforded by the slit lamp is sufficient, and therefore the Nernst slit lamp was used in my reflexless ophthalmoscopes. The large ophthalmoscope brought on the market by Carl Zeiss contains an aspheric lens not only in the examination system, but also in the illuminating system. The light of the latter is reflected into the examined eye by a transparent glass prism of small angle. A plate with parallel faces would give two different images, of which only one could be used if the plate were sufficiently solid. Therefore the prism is so calculated that both images coincide. In this instrument the Nernst lamp can be directly replaced by a nitra lamp or by a pointolite lamp. I have verified that the latter affords a sufficient illumination though the lamp be mounted along the axis of the illuminating system, and although only a part of the optical image of the glowing ball can be projected in a part of the slit.

The same is the case with the large instrument for *simplified reflexless ophthalmoscopy*. If two small reflexes caused by the surfaces of the aspheric lens are allowed, the construction of the instrument is

considerably simplified and only one aspheric lens is necessary. The light emerging from the slit is given the appropriate direction by means of a double reflection in a rectangular prism silvered on its hypotenuse face and combined with a decentrated lens.

For this method of ophthalmoscopy I also have devised a *hand ophthalmoscope*, which is held in one hand, the aspheric lens being held in the other. The illuminating system is a slit lamp in which a small incandescent lamp is used as the source of light. Again this instrument demands a little skill, which must be acquired by practice. Ordinary ophthalmoscopy by the indirect method should be replaced by this method just as well as ordinary ophthalmoscopy by the direct method is to be replaced by simple centric ophthalmoscopy.

CENTRIC PHOTOGRAPHY OF THE FUNDUS

It is evident that the same principles that led to the centric reflexless ophthalmoscopy are applicable theoretically to the photography of the fundus. In practice difficulties arise from the necessary intensity of light, and as long as perfectly aplanatic condensers of sufficient size and aperture are not available, the sun is the only practicable light source. But if an optical image of the sun is projected in a hole, the method of the simplified reflexless ophthalmoscopy is applicable and has been elaborated by Dr. J. W. Nordenson, who has obtained excellent results with a rather compendious apparatus. Thus the light illuminating the fundus for photography is taken from what could be named a hole lamp with the sun as source of light.

DISCUSSION

DR. EDWARD JACKSON (Denver, Col.): The name of Professor Gullstrand is universally known to us, but it will have a new significance after hearing the address he has given us this morning. We are especially happy in having in it a sort of summing up of the work he has been publishing for so many years, and that has made him so well known throughout the world. He has followed the normal method of instruction, starting with a concrete case with the slit lamp, and now bringing before us the general principles that were embodied in that, and which can be embodied in the use of light from other sources, the whole group to be called "diaphragm lamps."

Vision depends upon getting from the object to be seen a sufficient light, as compared with the light received from sources in which we are not interested. We are just beginning to realize in our methods of examination that, if we want enough light from the structure that we desire to examine, we must exclude irrelevant light in so far as this is possible. That has been accomplished best by the slit and other diaphragm lamps. The conditions of dis-

tinet vision that have to be met—that the light shall be bright enough (and external light is worse than useless) and that the field to be studied shall be uniformly illuminated. All of us have been misled for an instant by irregular illumination into thinking that we had some retinal or choroidal pathologic condition that was not present. That is accurately brought out by the use of the uniform illumination.

But in order to get rid of the light that we do not want we must have sharp limits, we must not light up anything except the structures we are looking at. That is obtained with the diaphragm lamp, by the diaphragm primarily, and then by having it sharply focussed in the eye. That is what Professor Gullstrand has worked out. First, he gave us enough light with the Nernst slit lamp, and then he had a sharp image of the slit formed in the eye where the light would be limited to a certain field.

Of course that can be done with the hole lamp, but I cannot help but feel that the slit offers certain advantages. As he has pointed out, we can still have the slit with the "hole lamp." This, I take it, is the idea running through the focussing apparatus that is worked out, and the result of utilizing the different methods of illumination. I have been interested in one phase of illumination, direct sunlight, and hope soon to call attention to the practical application of the principles that have been put before us by Professor Gullstrand in that direction.

DR. HARRY S. GRADLE (Chicago, Ill.): It might be of interest to speak of some few things that we have been endeavoring to do in this country. The Pointolite lamp was developed in England in 1913, and has been used rather extensively by microscopists and metallurgists, and in this country in microphotography with great success. For nearly a year it has been in our mind to employ the Pointolite with the apparatus for illumination of the eye for which we are indebted to Professor Gullstrand, the slit lamp, but owing to certain difficulties of material and manufacture it has been impossible until recently. Now we have a large tungsten arc in a low pressure gas chamber, giving as nearly a punctate source of light as possible. This may be used in the slit lamp, or it may be used when the slit is replaced by the diaphragm, resulting in a more nearly true focal illumination than the present slit. True it is that the slit has certain advantages for both direct and indirect focal illumination, but there are areas of aberration projected by the slit with the Nitra lamp on the cornea which increase as we narrow the slit down to a minimum, and which cannot be eliminated owing to certain mechanical difficulties. I believe if we could have a circular aperture with the Pointolite we would gain a great deal.

The large ophthalmoscope of Professor Gullstrand, unfortunately, does not add any to the clinical examination of patients in any but the very exceptional case, but to the ophthalmologist who takes pleasure in clean-cut, scientific work, it is a source of pleasure. The small hand ophthalmoscope of Gullstrand, which we have been using for eight or nine years, employs a slightly different principle, in which the lower portion of the cornea and of the pupil is illuminated and the rays returning from the fundus pass out through the upper portion of the cornea, which is without any illumination whatever

and consequently is reflex free. It makes indirect ophthalmoscopy much simpler and easier than by the older methods and allows us to study the macula without the corneal reflexes we are accustomed to.

DR. E. E. BLAAUW (Buffalo, N. Y.): On the question of the slit lamp I think the Congress stands where ophthalmology stood in 1851 when the ophthalmoscope was introduced. The lamp of Professor Gullstrand has given us a field of light so enormous that those who have not used the corneal microscope cannot appreciate what it means to us as ophthalmologists. But one should not say that its use is easy, for it takes hours and hours of study. We must be able to narrow the slit, because a slit that cannot be opened and closed is of no value. It is still difficult to interpret what we see with the slit lamp. Of enormous value is the appreciation of depth, for it enables us to see changes that have not been known before, and it shows us that we have no way of attacking the vitreous unless we do it *in vivo*.

PROFESSOR G. F. ROCHAT (Groningen, Holland): I have no doubt that the new instrument that Professor Gullstrand has spoken of will be a much more beautiful instrument than his former one, but I think the slit lamp in its previous form has one special advantage over the new lamp he has described, and that is this: When we throw a beam of light into the cornea, its intersection with the cornea is a rectangular prism, of which the anterior surface coincides with the anterior surface of the cornea, while its lateral side shows a definite rectangular and plane section of the cornea, and in this luminous section you can observe and localize particles present as clearly as in microscopic section. I am afraid that in the new form, in which the intersection of the cornea and beam of light is not plane, but cylindrical, this advantage will have been lost. I shall be glad to have Professor Gullstrand's opinion on that.

PROFESSOR ALLVAR GULLSTRAND (closing): I am surprised that so many of my American colleagues have devoted so much time to the study of my methods, and I want to thank those who have taken part in the discussion. Dr. Jackson has pointed out that you can get a good result with direct sunlight, and if I understood him aright he is operating with direct sunlight. I hope that the photographs that Dr. Nordenson will show tomorrow will prove that Dr. Jackson is right in trying sunlight. But I hope that the Pointolite lamp will be shown to be sufficient for most purposes.

Dr. Gradle spoke of the scientific work that can be done with the slit lamp, and he as well as Dr. Blaauw questioned the necessity of having a slit for localization in opacities, or in pathologic signs, as to the dimensions. We now have a beam of light that is not too wide. I think the Pointolite is the best in practical work.

SINTOMAS OCULARES DEL ENVENENAMIENTO POR EL PIQUETE DE ALACRÁN¹

DR. JOSÉ DE JESUS GONZALEZ

De Leon, Gto., Mexico

En la invitación dirigida por la Comisión General de Organización del Congreso Internacional de Oftalmología, que se reunirá en Washington, indícase que “son apropiados a los fines del Congreso los trabajos que se referan a enfermedades o fases de enfermedades, peculiares a los distintos países o regiones.”

Esta indicación me hace dar a conocer los *síntomas oculares* que he observado en personas que han sufrido el *piquete del alacrán*, algunos de los cuales síntomas, por persistir cuando todo otro síntoma de envenenamiento ha desaparecido y aun revestir cierta gravedad, son interesantes de conocer por todo oculista que ejerza en las regiones infestadas por los *escorpionídeos*.

Además, desde el punto de vista puramente científico, algunos de los síntomas oculares son altamente interesantes, porque contribuyen a precisar la acción de la ponzoña del alacrán.

Para la mejor inteligencia del asunto, divido este pequeño estudio en tres partes:

I. Algunos datos sobre los alacranes de México.

II. Sintomatología general del envenenamiento por el piquete de alacrán.

III. Síntomas y complicaciones oculares de dicho envenenamiento.

I. ALGUNOS DATOS SOBRE LOS ALACRANES DE MÉXICO

Los alacranes o escorpiones son *artrópodos*, pertenecientes a la clase de los *arácnidos* y al orden de los *escorpiones*, orden que comprende de 350 a 400 especies diseminadas por todo el mundo.

En la República Mexicana, según la Biología Centrali Americana, citada por el Sr. Moisés Herrera,² únicamente viven dos especies de la

¹ Estudio presentado al *Congreso Internacional de Oftalmología* que se reunirá en Washington, E.U.A., en Abril de 1922.

² Moisés Herrera, Naturalista de la Dirección de estudios biol.—Los escorpiones de México. Mem. de la Soc. Científica, “Antonio Alzate,” Tomo 39, México, 1921.

familia *scorpionidae*: el *diplocentrus whitei* (o *diplocentrus mexicanus*) y el *diplocentrus keyserlingi*, Karsch.

Pero el distinguido Profesor Isaac Ochoterena¹ recuerda que en la magistral obra *Das Tierreich* (8. Lieferung. Arachnoidea. 1899) los escorpionideos se dividen en seis familias, la primera de las cuales, la de los *Bothruridae*, comprende las familias *Centrurinae*, con los géneros *Centrurus*, *Isometrus*, *Zabius* y *Tytius*.

Los alacranes del género *Centrurus* (Hemprich u. Ehrenberg) comprende 14 especies, de las cuales se encuentran en la República Mexicana las siguientes: *Centrurus infamatus*, C. L. Koch, *Centrurus gracilis*, Latr., *Centrurus margaritatus*, Gerv., *Centrurus infamatus*, var. *nigrovariegata*, Poc., *Centrurus nigrimanus*, *Centrurus fulvices*, Poc., *Centrurus flavopictus*, Poc., *Centrurus ochraceus*, Poc., *Centrurus nitidus*, Thor.

Uno de los alacranes más venenosos del país, el conocido con el nombre de alacrán de Durango, (y que no es exclusivo de esa ciudad, pues, según el Dr. Mariano Herrera,² existe en todo el Sur del país, especialmente en las vertientes de la Sierra Madre) que ha sido bien estudiado por el Sr. Prof. I. Ochoterena, pertenece a la especie *Centrurus exilicauda*, descrito por Wood.

En la ciudad de León, Gto.,—en donde ejerzo desde hace 25 años, hay dos especies: una, poco venenosa, de color moreno oscuro (*diplocentrus mexicanus*?), y otra muy venenosa, de color amarillento, que tiene gran semejanza con el *Centrurus exilicauda*, de Durango. He oído decir a médicos que tienen muchos años de ejercer en la ciudad, que antes no observaban accidentes serios con el piquete del alacrán, lo que me hace suponer que tal vez ha sido importado el alacrán venenoso de Durango, oculto entre las pieles sin curtir que la industria peletera de la ciudad hace venir de las regiones del norte del país, entre ellas de donde abunda el escorpionideo.

Aunque suele encontrarse el alacrán durante todo el año, los meses en que más abunda en esta ciudad, sin duda por ser su temperatura media más favorable al arácnido, son los de marzo, abril, mayo y junio, durante los cuales, además, parece ser más activa la ponzoña, pues los accidentes que he observado entonces son más serios. Igual

¹ Prof. Isaac Ochoterena. El alacrán de Durango (*Centrurus exilicauda*, Wood). Memorias y Revista de la Sociedad Científica "Antonio Alzate," Tomo 37, núms. 4, 5 y 6, México, 1920.

² Dr. Mariano Herrera. Estudio sobre el piquete de alacrán. Revista Médica, Tomo XIII, núm. 1. México, 1900.

cosa pasa en Durango, según el Dr. Mariano Herrera, quien textualmente dice: "casi exclusivamente se observa a fines de abril, en mayo y en junio, terminando con los primeros aguaceros formales que se verifican en julio." Y agrega: "no porque no pueda haber accidentes en otras épocas del año, como se ve en los cuadros de mortalidad, sino porque tales accidentes tienen el carácter de excepcionales." (Loco citato.) La temperatura media de esos meses escila entre 19° y 25°.

El *aparato venenoso* consta de dos glándulas, colocadas a uno y otro lado del último segmento del post-abdomen. Cada glándula está constituida por un tejido conjuntivo periglandular, muy delicado, una gruesa capa de tejido muscular, cuya contracción expulsa el veneno, y el epitelio glandular con numerosas invaginaciones, capa propiamente secretante. De cada glándula parte un canal eferente que va a desembocar cerca de la extremidad del aguijón, que es curvo, muy duro, acerado y terminado por una punta roja. (Ochoterena.)

El *veneno* es líquido, transparente, de reacción ácida, propiedades que he observado personalmente. El Dr. Mariano Herrera cree que es de naturaleza oleoginosa, pues haciendo picar a un alacrán sobre un papel delgado, se obtiene una pequeña mancha translúcida y persistente, análoga a la que deja un líquido aceitoso. El Prof. Ochoterena dice que se altera rápidamente al contacto del aire, que, disuelto en agua, se vuelve opalescente con irisaciones azuladas y en parte precipita (globulinas), produciendo espuma si se agita. El alcohol, el yodo, el amoníaco, el tanino, el nitrato de plata, el acetato de plomo y el sulfato de amoníaco, lo precipitan de sus disoluciones y el producto de la evaporación en el vacío está formado de laminillas de color amarillo obscuro.

Parece que esta ponzoña es una toxi-albúmina especial (Calmette). Su actividad es extraordinaria, pues la cantidad introducida por un piquete, es muy pequeña: inmediatamente despues de producida en mí mismo una picadura en un dedo, he podido extraer, por expresión fuerte, una pequeña gota de poco más de un milímetro de diámetro.

La manera de obrar la ponzoña, nos la va a decir el cuadro sintomático desarrollado a consecuencia del piquete de alacrán.

II. SINTOMATOLOGÍA GENERAL DEL ENVENENAMIENTO POR EL PIQUETE DE ALACRÁN

Para que sirva de punto de comparación a los observadores de otras regiones, voy a desarrollar el cuadro del envenenamiento por la ponzoña de alacrán, basado exclusivamente en lo que he observado personalmente.

El piquete produce la sensación de una delgada aguja que rápidamente se hundiera en la piel; pero esta dolorosa sensación—que he experimentado en mí mismo—desaparece casi inmediatamente y por algunos minutos se cree que nada va a pasar. Y en efecto: si se exprime *inmediatamente* el punto picado, sale una pequeña gota del líquido venenoso, y no se produce ningún otro síntoma, ni local ni general. Para facilitar la expresión del veneno, es conveniente practicar una pequeña incisión de la piel; desgraciadamente es raro que esta pequeña operación se haga oportunamente.

Entonces se deja que el veneno se absorba, para lo cual sigue dos caminos: el de los capilares linfáticos y el de los capilares sanguíneos. Pocos minutos después comienza una sensación de *hormigueo* en las partes cercanas al piquete y pronto esa penosa sensación se generaliza, siendo uno de los síntomas que más torturan al enfermo. Algunos de ellos me han comparado ese hormigueo al que se sufre cuando empieza a volver la sensibilidad a un pie que se ha *adormecido* por la compresión del ciático. Otros dicen que son finas y repetidas corrientes eléctricas que circularan por todo el cuerpo, inclusive por las vísceras abdominales: un enfermo permanecía inmóvil, rogando que no se le tocara, porque una corriente eléctrica parecía atravesarle los intestinos.

En la cara, principalmente en los párpados y en las alas de la nariz, experimentase un cosquilleo insoportable, que origina penosos y frecuentes estornudos.

Al lado de estas *parestias cutáneas*, señalaré uno de los síntomas más frecuentes y más precoces: *la sensación de una maraña de cabellos en la lengua y en la garganta*. Si se permite la extensión del término, diré que es esta una *parestesia mucosa*, ya que parestesia es toda sensación *subjetiva* anormal.

También existen perturbaciones *objetivas* de la sensibilidad: la *hiperestesia* es intensa; cualquier contacto, el más ligero frotamiento, causan al enfermo sensaciones dolorosas o de quemadura y aun despiertan intensos reflejos cutáneos o musculares.

Creo que debe atribuirse a la hiperestesia de la mano y de los dedos una perturbación de la *percepción estereognóstica*: los enfermos reconocen bien todos los objetos por el tacto; pero les conceden dimensiones mayores a las reales. Podría designarse esta perturbación con el neologismo de *disestereognosia* o, haciendo una contracción: *distereognosia*.

Simultáneamente con las perturbaciones sensitivas, desarróllanse otras *secretoras* y *motrices*.

Entre las *secretoras*, la hipersecreción saliver, de una saliva glutinosa y espesa, y de las mucosas nasal, faríngea y brónquica, constituye uno de los síntomas más precoces, que indica que la ponzoña se ha generalizado y van a empezar los fenómenos convulsivos de que luego hablaré. El enfermo se ve obligado a estar continuamente escupiendo y expectorando, sin conseguir amenguar la penosa sensación de cabellos que se le enredaran en la garganta.

Señalaré en este lugar, por tratarse de una perturbación secretora, aunque el síntoma se presente más tarde, la hipersecreción, de sudor que es abundantísima: toda la piel del enfermo está constantemente bañada en sudor. Tal parece que el organismo escoge por *vias de eliminación del veneno*, el sudor, la saliva y las secreciones nasofaríngeo-brónquicas.

Son las *perturbaciones motrices* las más penosas y las que ponen más en peligro la vida del enfermo. Se presentan pocos minutos después de que el hormigueo se ha generalizado y consisten en *espasmos de los músculos lisos* y *convulsiones y contracturas de los voluntarios*.

Los *espasmos* empiezan por la *faringe* y el *esófago*: los enfermos, atormentados por la sed, se sorprenden de que no pueden pasar el agua o lo hacen con suma dificultad. Los espasmos del *estómago* producen vómitos, primero alimenticios y luego mucosos. Aunque raros, hay casos en que contrayéndose el estómago en vacío, se producen hemorragias y hematemesis de cierta gravedad. Los espasmos del *intestino* dan a los enfermos la penosísima sensación de que una corriente eléctrica atraviesa todos los órganos abdominales. El espasmo del *cuello vesical* origina retención de orina, la que, por otra parte, es escasa, probablemente por la abundante sudación. Los espasmos de los músculos brónquicos contribuyen sin duda a aumentar los fenómenos asfíxicos de que pronto hablaré.

Las *contracturas* se presentan principalmente en los maseteros, originando un *trismus* que dificulta la ministración de medicamentos *por ore*.

Las *convulsiones* pronto se generalizan y agitan contorcionan todo el cuerpo. Son de caracter *tónico*, a veces los músculos se tetanizan y cuando el tetanismo invade los músculos respiratorios, se presenta la asfixia, exagerada por la abundante hipersecreción brónquica y los espasmos de los músculos lisos de la par red del arbol aereo. En los niños estos accidentes son a menudo mortales: la respiración es estertorosa y superficial, los labios se cianosan, por la boca salen mucosidades espumosas, frecuentes accesos de tos convulsiva interrumpe y agrava el tetanismo de los músculos respiratorios, un sudor frio empapa los cabellos del niño y corre por el cuerpo, y el pequeñuelo cae en un estado semicomatoso en el que a veces sucumbe. Pocas veces en el adulto llega la gravedad a tal extremo, aunque también hay casos mortales.

En el niño he observado convulsiones del diafragma y de otros músculos respiratorios que se traducen por un llanto espasmódico, tan característico, que en dos ocasiones, con sólo oír de lejos ese llanto en niños que eran conducidos a mi consultorio, pude hacer el diagnóstico de piquete de alacrán, luego confirmado por los padres del pequeño.

En el adulto, en los momentos de mayor intensidad del envenenamiento, las convulsiones tónicas agitan y contorsionan los brazos y las piernas, las mucosidades excitan la laringe y producen accesos de tos y expectoración, el tialismo es abundante, un sudor copioso empapa todo el cuerpo, la temperatura se eleva hasta 40° y 41°—tanto por el exceso de trabajo muscular, como por acción de la ponzoña sobre los centros termógenos, pues a veces las convulsiones son poco intensas y sin embargo la temperatura asciende-, el enfermo siente un calor sofocante y, presa de grande inquietud, no encuentra sitio en su lecho para reposar un instante, y grita y gesticula y se retuerce. El pulso es frecuente.

La cara está congestionada y edematosa.

La *palabra* es ininteligible: el trismus, las contracciones de los músculos de la lengua, la abundancia de saliva y de mucosidades, la horrible sensación de la maraña de cabellos, son todas circunstancias capaces de hacer la palabra tartajosa e ininteligible.

Por regla general, la *inteligencia* se conserva, en medio de aquel cuadro intensamente dramático: el enfermo entiende nuestras preguntas y contesta a ellas con acierto. Pero aun entonces el psiquismo no es enteramente normal: la atención, prisionera del intenso malestar, no puede fijarse en nada; las emociones exaltativas, sobre todo la

ira, contribuyen a aumentar la exaltación del enfermo; la memoria es torpe. Pero no hay alucinaciones, ni ilusiones, ni delirio. Solo en los casos graves y fatales, el enfermo cae en un estado de estupor y de inconciencia, precursor de la muerte.

Todo este *período*, que debe llamarse conjusticia, *de excitación*, es variable en intensidad y en duración, según diversas circunstancias: edad de la persona picada, sexo, época del año, cantidad de ponzoña inyectada; pues los niños, los ancianos y las mujeres son más susceptibles, los meses de marzo a junio, los de mayor actividad del arácnido, y las horas que siguen al crepúsculo las más peligrosas, antes de que el alacrán haya tenido tiempo de picar otros objetos—insectos, etc.—y, por lo mismo, antes de que haya vaciado en parte sus glándulas.

El *período de excitación* dura de 12 a 24 horas y los síntomas se van poco a poco atenuando y disminuyendo la gravedad de la situación.

Pero al período primero sigue otro *período de depresión*: a las convulsiones, suceden la torpeza en los movimientos, los temblores, la ataxia y aun verdaderas parálisis, que suelen durar unos días: he visto un enfermo que conservó por una semana cierta incoordinación motriz, otro que sufrió una paraplejia, una pobre anciana que quedó con una parálisis de la vejiga que no llegó a ceder, pues exigiendo el frecuente sondeo, acabó por infectarse la vejiga y producirse una cistitis mortal.

A los hormigueos del principio, siguen sensaciones de adormecimiento, sobre todo en las extremidades; es decir: a las *parestias de excitación* suceden *parestias de depresión*.

Todavía días después, la deglución se hace con dificultad, pero no ya por espasmo, sino por paresia faringea.

Condensando en una *clasificación* todos los síntomas observados, tenemos:

Síntomas sensitivos: dolor, en el lugar de la inyección, *parestias* (hormigueos, cosquilleos, sensación de maraña de cabellos), *hiperestesias*.

Síntomas secretorios: hipersecreción de las glándulas salivares, mucosas de la nariz, faringe y broquios, glándulas sudoríparas.

Síntomas vaso-motores: vaso-dilatación de los vasos de la piel de la cara, de la mucosa nasal, de la conjuntiva; edema de la cara.

Síntomas térmicos: hipertermia en el período de excitación; hipotermia en los casos fatales.

Síntomas motores: En el período de excitación, *espasmos* de los músculos lisos; *contracturas* y *convulsiones* de los músculos estriados.

En el período de depresión: músculos lisos, *paresias*; músculos estriados, *temblores*, *ataxia*, *parálisis*.

Reflejos: período de excitación, *exagerados*; período de depresión, *normales*.

Se ha dicho que mientras el veneno de la serpiente es neurotóxico, el del alacrán es miotóxico. El análisis cuidadoso de los síntomas que acabo de describir, nos inclinarán más bien a considerar la ponzoña del alacrán como *neurotóxica*, llevando su acción *excitante* sobre la médula (exageración de los reflejos, fenómenos motores, fenómenos sensitivos), sobre el simpático (vaso-dilatación, edema, espasmos de los músculos lisos, taquicardia, hipertermia, hipersecreción) y aun sobre los cordones nerviosos mismos, como parecen demostrarlo algunas complicaciones oculares que luego describiré.

A propósito de la hipersecreción salivar, debo recordar algunos datos recientes sobre la fisiología del simpático: las glándulas submaxilares y sublingual, que son las directamente influenciadas con la excitación del simpático cervical, producen una saliva *viscosa*, es decir, exactamente igual a la que se produce en los emponzoñados con el piquete de alacrán. Las parótidas, cuya saliva es espumosa y más fluida, *no son directamente influenciadas* por la excitación del simpático. Estos datos hablan en favor de la acción excitante, sobre el simpático, del veneno del alacrán.

El veneno del alacrán es, pues, *neurotóxico excitante*; los fenómenos de depresión que se encuentran después que cesaron los fenómenos tormentosos, pueden ser atribuidos a agotamiento de la celdilla nerviosa, que ha hecho tan fuertes gastos de energía.

No quiero terminar sin decir dos palabras sobre *terapéutica*. Mientras no poseamos un suero específico como el anti-cobraico, como el anti-crotálico, nos veremos reducidos a dos medios curativos: uno, la eliminación del veneno; otro, la medicación sintomática. Para *eliminar el veneno*, tenemos que recurrir a las vías indicadas por la naturaleza: el sudor y la saliva. El medicamento indicado es la *pilocarpina*, en inyecciones hipodérmicas, vigilando su empleo: en mis manos ha sido muy eficaz. La medicación *sintomática* debe dirigirse principalmente contra el síntoma más peligroso: los espasmos respiratorios. Las *inhalaciones de cloroformo*, hechas con la mayor prudencia, nos darán tiempo para esperar la eliminación del veneno, único medio de salvar la vida del enfermo.

III. SÍNTOMAS Y COMPLICACIONES OCULARES DEL ENVENENAMIENTO POR EL PIQUETE DE ALACRÁN

Durante el período de excitación, en medio de la situación tan dramática y ante la gravedad del pronóstico *quoad vitam*, los síntomas oculares ocupan un segundo lugar ante la atención del médico, aunque a veces molestan mucho a los enfermos; pero, pasados los momentos de peligro y de angustia, las manifestaciones oculares del envenenamiento empiezan a ocupar un lugar preponderante y, en ocasiones, llegan a constituir verdaderas complicaciones que, habiendo desaparecido todo otro síntoma, son ellas la única enfermedad. Salta a la vista la importancia que entonces alcanzan para el oftalmologista.

En el primer período del envenenamiento—período que he llamado de *excitación*—los síntomas que se presentan del lado de los ojos son de toda clase: *sensitivos, secretorios, vaso-motores, motores y sensoriales*. Los señalaré en este orden.

Perturbaciones sensitivas.—Una de las regiones en que son más intensas las parestesias que he señalado, es en la ocular: los párpados y la conjuntiva son el sitio de hormigueos y cosquilleos molestísimos, que obligan a los enfermos a frotarse continuamente los ojos, cuando menos al principio, antes de que las intensas convulsiones les impidan hacerlo.

Perturbaciones secretoras.—A la vez que las hipersecreciones de que he hablado, se establece un abundante lagrimeo. Precisa recordar que si la secreción lacrimal es *principalmente* producida por la acción de las fibras orgánicas del sistema craneal (para-simpático craneal), también la provoca la excitación del simpático cervical; la hipersecreción salivar viscosa, como la que produce la excitación del simpático cervical, me hacen pensar que también el lagrimeo está bajo la dependencia de la excitación de esta parte del simpático, aunque no se limita a ella la acción de la ponzoña, como lo indican síntomas tales como los espasmos intestinales y la contractura del esfínter del cuello vesical, que se deben a la excitación de la cadena lateral lobar, para no citar otros.

Perturbaciones vaso-motrices.—Dos fenómenos de este orden podemos señalar: la hiperemia de la conjuntiva y el edema de los párpados. Es muy difícil explicar la vaso-dilatación de los tegumentos de la cara y de las mucosas, que es un síntoma de interrupción del simpático cervical, al lado de otros síntomas de excitación, como las hipersecreciones lacrimal, salivar, sudoral. ¿Se trata de acción directa de

la ponzoña del alacrán sobre las fibras musculares de la pared vascular? O bien la intensa actividad glandular en la piel y las mucosas, provocada por la excitación del simpático, tendrá como consecuencia la producción de *metabolitos*—según la teoría de Gaskell—que tendrían una acción vaso-dilatadora? No puedo hacer más que emitir estas hipótesis.

Perturbaciones motrices.—Prodúcense tanto en los músculos extrínsecos, como en los intrínsecos del ojo. En el período de excitación—que es el que describo en este momento—he observado variados estrabismos, predominando el convergente, debidos a *contracturas de los músculos óculo-motores*.

Del lado de la musculatura *intrínseca*, el síntoma más constante es la *miosis*. Habiendo otros síntomas de excitación del simpático, la miosis no podría ser atribuida a la parálisis de este sistema, tanto más cuanto hay fenómenos de excitación del motor ocular común—que acabo de señalar—y que explican perfectamente la miosis por contracción del esfínter. Para precisar mejor esto, he recurrido, como medio de diagnóstico, a las instilaciones de solución de cocaína: recuérdese que en la miosis por parálisis del simpático (parálisis de las fibras dilatadoras de la pupila) la instilación de cocaína produce midriasis, mientras que queda sin efecto en la miosis por excitación del motor ocular común (contracción del esfínter pupilar). Esto último pasa en la miosis de los emponzoñados por el veneno del alacrán.

A la miosis acompañan espasmos de la acomodación que, provocando miopía acentuada, son una de las causas de la perturbación de la vista y tal vez de la micropsía de que se quejan algunos enfermos.

Perturbaciones sensoriales.—Quéjense los enfermos de que una niebla envuelve todos los objetos y de que las luces están rodeadas de círculos irisados; estos síntomas y la fotofobia, que casi nunca falta, podrían ser atribuidos a la acción de la ponzoña sobre la retina, directamente, como parecen demostrarlo algunas observaciones, o a la congestión retiniana que probablemente se encuentra, ya que la vasodilatación se extiende a todos los vasos cutáneos y mucosos, pero que no he podido comprobar al oftalmoscopio, por impedírmelo la miosis y la agitación motriz del enfermo.

La diplopía, causada por el estrabismo y la micropsía, seguramente consecutiva al espasmo de la acomodación, son nuevos factores que contribuyen a la producción de las perturbaciones visuales.

Por regla general, todos estos síntomas van atenuándose a la vez

que los síntomas generales, y el enfermo, al recobrar la salud, no conserva ninguna perturbación del lado de sus ojos.

Pero no sucede siempre así: a las contracturas de los músculos extrínsecos o intrínsecos del período de excitación, siguen *paresias* más o menos acentuadas y hasta verdaderas *parálisis*, y como estas suelen persistir no solamente días, sino hasta semanas, puede pasar inadvertida para el oculista la verdadera causa que las produce, cuando el enfermo no da enseñanzas sobre el particular.

Personas completamente sanas de sus ojos y que no se quejaban de ninguna perturbación visual antes de sufrir el piquete del alacrán, vienen al consultorio a contarnos que, desde que lo sufrieron, no pueden ver como antes, que los objetos lejanos les parecen borrosos o que les es imposible leer. El examen descubre, unas veces, parálisis de los músculos extrínsecos, en ocasiones tan leves, que no provocan desviación de los ejes oculares y sólo pueden ser descubiertas por el examen con los vidrios coloridos, que hacen aparecer la diplopía; otras veces, lo único que hallamos es la parálisis de la acomodación que nos obliga a prescribir al enfermo—mientras por otros medios la hacemos ceder—el porte de cristales de $+ 1.50$ D. a $+ 2$ D., para los trabajos de cerca.

Debo advertir que he tenido oportunidad de observar el cuadro dramático del envenenamiento, en personas perfectamente sanas de sus ojos, y en las que, después, he encontrado las parálisis y las paresias de los músculos oculares que acabo de señalar, así es que no abrigo duda sobre esta acción del veneno del alacrán.

Pero si estas complicaciones no carecen de importancia, mucha mayor la tienen las que voy a describir en las observaciones siguientes, tanto porque en estos casos la *ceguera absoluta* persistió por algunos días, como porque parecen demostrar que el veneno del alacrán obra directamente sobre los cordones nerviosos, pues, en uno y otro caso, el oftalmoscopia permitió ver alteraciones en el nervio óptico.

Obs. I.—*Edema ligero del nervio óptico, con amaurosis completa, consecutivo a piquete de alacrán.*—Sra A. N., de 50 años de edad, sin antecedentes morbosos. Sufre al piquete de alacrán en una mano, estando en perfecta salud; en seguida se desarrolló todo el cuadro del envenenamiento que he descrito—hormigueos, sialorrea, sudor abundante, convulsiones, espasmos, etc.—revistiendo media intensidad. Desde el principio note la enferma que se nublaba su vista, pero esperaba que, al pasar los efectos de la ponzoña, recobraría su buena vista habitual: no fué así, sino que la ceguera se acentuó hasta llegar a ser completa.

Al día siguiente del piquete, el examen ocular me reveló la completa normalidad en el exterior de ambos ojos, así como la integridad de todos los movimientos del globo. Ambas pupilas estaban dilatadas y no respondían al estímulo luminoso, ni a la convergencia. *Agudeza visual, nula.* Por el examen oftalmoscópico encuentro los medios transparentes; pero *las dos papilas* estaban ligeramente *edematosas*: contornos borrosos, mayor diámetro que el habitual, venas más gruesas que normalmente. Ningún otro síntoma oftalmoscópico. Tensión normal en los dos ojos. Ningún dolor, ni espontáneo ni a la presión.

Atribuí el edema de los nervios ópticos a la única causa que me señalaban los antecedentes: la ponzoña del alacrán, y como la pobre enferma casi no había tenido asistencia médica durante lo agudo de su envenenamiento, recurrí a una inyección de cloruro de philocarpina, para eliminar las últimas cantidades de ponzoña; los resultados fueron favorables, pues la enferma empezó a ver. En los días siguientes prescribí 1 gr. diario de yoduro de sodio, cuya acción sobre el exsudado papilar me pareció eficaz, pues en una semana el aspecto del fondo del ojo se volvió normal y la enferma recobró completamente la vista.

Obs. II.—*Ceguera completa, con pocos síntomas oftalmoscópicos, consecutiva a piquete de alacrán.*—Una jovencita de 17 años, de buena constitución y en pleno estado de salud, sufre el 6 de diciembre de 1920, un piquete de alacrán en una mano, seguido de todos los síntomas de envenenamiento y de ceguera completa que persistió 24 horas después que había desaparecido todo peligro. No la observé entonces.

El 28 de diciembre del mismo año, es decir, a penas tres semanas más tarde, sufre nuevo piquete de alacrán en el antebrazo izquierdo. En esta vez los fenómenos de envenenamiento fueron mucho más intensos: trismus, espasmo faringeo, convulsiones tetaniformes, fenómenos asfíxicos y pérdida de conocimiento. La enferma estuvo luchando entre la vida y la muerte durante 24 horas. Al volver a la plena conciencia de sí misma, notó que *no veía absolutamente.*

No recurrió a mis cuidados sino hasta después de ocho días y cuando ella y sus padres habían ya perdido toda esperanza de que se recobrara la vista espontáneamente. Entonces encontré lo siguiente:

Ningún síntoma nervioso general.

Movimientos normales de los globos oculares y de los párpados.

Exterior de ambos ojos, normal. Tensión, normal.

Pupilas midriáticas—cinco milímetros de diámetro—y sin reaccionar a la luz.

Agudeza visual, *nula*: la enferma no distinguía la luz de la oscuridad.

Al oftalmoscopio encontré los medios transparentes; en el fondo del ojo únicamente se veían ambas papilas *muy rojas*, pero el calibre de los vasos era normal y los contornos papilares bien limitados.

La absoluta ceguera de la enferma, con tan pocas alteraciones oftalmoscópicas, me hizo pensar tanto en una neuritis retrobulbar—por la hiperemia papilar tan evidente—como en una acción *estupefaciente*, sobre la retina, de la ponzoña del alacrán.

La sudación provocada por una sola inyección de pilocarpina, y una serie de inyecciones de estricnina como tónico retiniano, fueron poco a poco mejorando a la enferma, que recobró su vista en el breve espacio de *doce días*.

Este caso es interesante por haberse presentado dos veces la ceguera en la misma persona, y de mayor duración en el segundo piquete, como si se hubiera producido anafilaxia.

DISCUSSION

DR. FRANCISCO M. FERNANDEZ (Havana, Cuba): As Colonel Elliot has well said in his book on "Tropical Ophthalmology," which I have just had the pleasure of translating into Spanish, the ocular lesions caused by the arthropods in the different tropical countries chiefly depend upon what part of the tropics they are observed, but are all much the same all over.

We have had no occasion of observing in Cuba the cases described by Dr. Gonzalez, perhaps because the scorpions we have there are not of the dangerous Durango variety identical to the *Centrurus exilicauda*; and their bites do not give rise to serious toxic symptoms, either general or ocular, but only to local manifestations.

The insectivorous life in Cuba is not abundant, and outside of the frequent cases of small flying insects that produce conjunctivitis, we can only remember now some cases of conjunctivitis of more serious nature produced by the toads that have been described by our teacher, Santes Fernandez, and that have presented a violent characteristic. These cases, however, have been produced by the local access of the secretion in the conjunctival sac and have not given rise to any general toxic manifestations.

DR. AURELIO BERAUN (Peru): Ocular lesions caused by the bite of the scorpion are rare in Peru. The paper is important and the symptomatology is well described. I have had, however, no experience.

COLONEL R. H. ELLIOT (London, England): I would like to take this international opportunity to direct attention to the fact that work in the tropical branches of ophthalmology has been very scanty. Very little indeed has been done, for instance, on the stings of scorpions and insects of various kinds.

May I start with the analogy of snake poisons? Some years ago I worked out the physiology of cobra venom. It was then generally assumed that if one had worked out one snake's venom, one had worked out the venom of all snakes; but I soon found that there was a difference between cobra venom and that of the other colubrine snakes (*e. g.*, the kareut and the sea snakes), and a much wider difference still between the venom of colubrine snakes and that of vipers. I believe the same thing is true of the stings of scorpions in different parts of the world, and that they are each specific in their action. Indeed, the natives of India will tell you that the little white scorpion's bite is worse than that of the large black one in the same district. We have heard today that an ophthalmologist working in one area has found definite symptoms after scorpion stings, while the rest of us have seen a large number of scorpion stings without any of these symptoms. I would suggest that it is a matter of the specificity of the poison in question.

After experimenting with snake poisons I worked a little with scorpion poison, and I was led to believe that the action of scorpion poison is closely allied to viper poison. Even here there are specific differences—the intense pain of scorpion sting is an illustration of this. I believe that scorpion poison runs along much the same line as the viperine venom, and I want to make two suggestions to Dr. Gonzales: (1) One of the characteristics of viperine poison is the clotting that occurs in the small vessels all over the body with hemorrhages following. It would be interesting to know whether in any of these cases of severe scorpion poisoning you get such hemorrhages. (2) Running right through all the snake poisons I have worked with is the tendency to vasomotor constriction. To test for this I perfused the vessels of the frog with a weak solution of various venoms, and regularly found a marked tendency to vasomotor constriction. May it not be that the blindness spoken of by Dr. Gonzalez as a result of scorpion poison is due to a special constriction of the retinal and choroidal circulation, an exaggeration of the general vasoconstriction which, as I have shown, runs through all snake poisoning? I would like to persuade men who are working in the tropics to take up these questions of the eye symptoms of snake, scorpion and allied poisons. I believe they will get a harvest of material from it.

DR. LLOYD MILLS (Los Angeles, Cal.): I had some experience in this subject in 1910. I was invited by the Southern Pacific Railroad Company of Mexico to investigate the reason why scorpions were holding up the construction of their road. Their road passes through some old, ruined Aztec villages, and when the adobe walls were torn down scores of these scorpions would run out, and the men were stung on their feet and hands. Twenty or thirty workmen were seriously ill. Five small children and two old people died. At that time no particular attention was paid to eye conditions, as our object was the development of an anti-venom. We took six puppies of the same weight and injected them with a mixed venom from the same type of scorpion, using an increasing dose, from 1 to 6. All of these animals were examined after four hours and at two-hour intervals, and we found the main objective condition to be an increasing meningeal congestion with an increased quantity of fluid in the meninges, to the point that I was led to believe that many of the general symptoms were due to a serous meningitis, though the neurotoxic symptoms

were the predominating ones. I would not be surprised if routine examination of the fundus in the graver cases would show various phases of a neuroretinal congestion with a distinct swelling of the disc in the more decided cases, the main source of these changes probably being found in the serous meningitis.

TRAITEMENT DU TRACHOME PAR LES INJECTIONS SOUS CONJONCTIVALES DE CYANURE DE MERCURE

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Paris, France

A défaut de substances réellement spécifiques dans le traitement du trachome nous possédons une foule de moyens médicaux et chirurgicaux pour arrêter l'extension du processus morbide, sinon pour le faire disparaître complètement. Une des conditions essentielles pour que les méthodes, d'ordre purement médical, soient efficaces c'est que leurs applications soient répétées pendant de longs mois et même des années. Les procédés chirurgicaux sont plus énergiques, plus satisfaisants et réduisent la durée du traitement. Malheureusement leur action est temporaire et ils ne sont pas sans inconvénients. Tout le monde connaît les cicatrices et les conséquences facheuses des caustiques violents et des scarifications trop étendues. De plus, employés seuls, ces procédés sont insuffisants à réaliser une cure radicale. Il fallait un procédé énergique, rapidement efficace et laissant le moins de séquelles possibles. Le problème nous semble résolu par l'emploi des injections sous conjonctivales de cyanure Hg, qui offre sur le jéquirity un avantage pratique, c'est d'être d'un dosage sûr et d'un maniement facile.

Bien que le virus ne soit pas encore connu, il est incontestable que cette affection est d'ordre infectieux; on a donc cherché, depuis bien des années, à exercer une action bactéricide. Tous les antiseptiques y ont passé même le cyanure. Mais si les lésions ont persisté, ou paru peu influencées, ce n'est point à cause d'une résistance particulière du virus, mais bien en raison du siège profond des lésions sous la muqueuse siège qui paraît les soustraire à l'action des antiseptiques employés superficiellement. Déjà les anciens s'étant rendu compte de ce fait avaient conseillé le massage, le raclage, le brossage ou les scarifications. De nos jours on préconise l'électrolyse ou la galvanocaustie selon la méthode d'Abadie. Ces méthodes peuvent être con-

sidérées comme satisfaisantes, mais nettement inférieures aux injections de cyanure.

Ce procédé n'est pas absolument nouveau. En Novembre 1920 deux Docteurs Roumains, MM. Lobel et Stiacovici en parlaient dans une communication publiée dans les Archives d'Ophtalmologie (de Paris). Ils le conseillaient dans les cas d'ulcères par suite de trachome. Leur technique diffère un peu de la nôtre. De plus ce procédé nous était parfaitement inconnu lors de nos premiers essais.

Travaillant à l'Hôtel-Dieu de Paris, dans le service de M. le professeur de Lapersonne, il nous a été donné de constater chez presque tous les malades à hypopion à qui nous faisons des injections sous conjonctivales de CyHg un épaississement de la conjonctive avec formation de tissu fibreux. Un genre de cicatrice se présentant sous la forme d'une plaque plus ou moins grande à l'endroit où avait eu lieu l'injection; ce tissu était plus ou moins transparent, blanc, jaunâtre, adhérent au tissu sous adjacent, parfois assez terne, conservant toujours cependant sa vitalité et sa sensibilité. Nous nous sommes demandés quel pouvait être l'effet d'un pareil traitement dans les cas de pannus trachomateux. Il nous a été permis de l'essayer sur un trachomateux qui se trouvait alors dans le service, (observation 1). Notre maître, qui suivait les malades, nous encouragea à continuer et nous guida dans notre étude par ses conseils bienveillants.

Aujourd'hui, bien que nos essais soient à leur début, nous croyons utile de faire part à Nos Confrères Ophtalmologistes de ce procédé contre ce véritable fléau qu'est le trachome. Nous espérons qu'ils contribueront au perfectionnement de ce nouveau procédé thérapeutique qui nous paraît devoir rendre service dans l'avenir.

TECHNIQUE

La technique est très simple: 1. Instillation de 4 gouttes de cocaïne à 4% à 1 minute d'intervalle. L'anesthésie faite, le malade est couché: cette position n'est pas absolument nécessaire, elle doit être choisie dans le cas où le malade est indocile ou pusillanime.

2. Si la fente palpébrale est rétrécie, par suite de processus inflammatoire, ou lorsqu'il existe du blépharospasme, on place un blépharostat. Dans le cas contraire et lorsque le malade est facile, il suffit d'écarter les paupières avec le pouce et l'index de la main gauche. On fixe la conjonctive avec une pince en prenant toute précaution utile pour ne pas la déchirer. On soulève alors légère-

ment la conjonctive qui fait un pli on enfonce rapidement mais prudemment l'aiguille d'une seringue de 1 cm³. On injecte lentement la solution suivante:

1. Solution.
Cyanure d'hydrargyre un milligr.
Eau distillée—un centimètre cube.
2. Solution.
Novocaïne—deux centigr.
Adrenaline au millième—deux gouttes.
Eau distillée—un centimètre cube.
Prendre dans la seringue de 1 c³, une partie de la solution N° 1 et une partie de la solution N° 2.

Une fois au courant de la technique des injections sous conjonctivales on fera mieux d'éviter l'emploi de la pince à fixation. De plus pour diminuer les risques d'ecchymoses, nous conseillons l'usage d'aiguilles très fines. Ceci diminuera les chances d'issue du liquide injecté, car il a tendance à ressortir dans les cas où il est, pour ainsi dire, sous pression par suite de l'hypérémie conjonctivale et de l'oedème inflammatoire, choses fréquentes dans le trachome aigu.

Si on procède lentement, le liquide se diffuse, soulevant la conjonctive en une boule qui contourne la cornée, sans avoir à repiquer et sans faire avancer l'aiguille. Il nous semble qu'en agissant ainsi on diminue la fréquence des ecchymoses en diminuant le nombre des vaisseaux lésés ou coupés au passage. Un pansement suffisamment compressif est appliqué pendant 12 à 24 heures selon la tolérance du malade. S'il arrive que le malade se plaint de quelques douleurs on desserre un peu le pansement. Quelquefois la suppression complète et définitive de la bande est la seule chose qui assure un soulagement. Souvent il faut avoir recours à l'aspirine.

En présence de douleurs parfois intenses chez les jeunes sujets nous avons pu les prévenir complètement, en administrant au malade O gr 50 d'aspirine une demi-heure avant, O gr 50 au moment même de la piqure et O gr 50 enfin deux heures après. Le soir même ou le lendemain on fait appliquer des compresses chaudes 3 fois par jour et des compresses froides les jours suivants. Cinq jours de repos et au sixième une nouvelle injection au même endroit ou du côté diamétralement opposé s'il y a lieu.

Comme inconvénients, à part la douleur, nous n'en connaissons pas qui contredisent l'emploi de cette méthode. Et encore avec l'usage d'aspirine arrive-t-on à prévenir toute douleur. D'ailleurs elles diminuent progressivement mais rapidement après chaque piqure.

Est-ce là un phénomène d'accoutumance de la conjonctive? Nous sommes plutôt enclins à voir la cause dans l'amélioration de l'état général du sujet et de l'état local de la conjonctive après les premières injections. Ce qui confirme cette idée c'est le peu de réaction chez les personnes dont les yeux sont peu enflammés, comme dans les cas récents de plaies pénétrantes du globe.

Un autre moyen nous a paru très efficace contre les douleurs; c'est les compresses chaudes qui, si elles sont appliquées quelques minutes après l'injection, réduisent à rien la réaction et les douleurs.

En suivant cette technique, on n'a pas d'hématome et d'ecchymose sous conjonctivale, peu esthétiques, et assez fréquents dans les cas de pannus. Si on tient compte de la solution de cyanure: 1:2000 on évite les eschares et les cicatrices qui en résultent.

RESULTATS

Au moment même de l'injection, si la solution est bien faite et la conjonctive bien anesthésiée, le malade ne sent absolument rien. Une demiheure après apparaît une gêne, se traduisant par une lourdeur générale de la tête des battements dans les tempes, et une sensation de douleur dans la région oculaire dont l'intensité va en augmentant jusqu'à une heure et demie après l'injection, pour diminuer petit à petit et disparaître complètement 3 heures après. Si on a eu soin de donner au malade 0 gr 50 d'aspirine au moment de l'injection, les phénomènes subjectifs se réduisent à ce qu'un de nos malades à essayer de nous décrire "Je ne souffre pas mais mon oeil est tiraillé." Si on enlève alors le pansement et qu'on examine le malade, on constate un oedème hypérémiq ue des paupières celles-ci deviennent souvent très volumineuses et la tuméfaction descend parfois jusqu'à la joue. Tous les tissus paraissent imbibés jusque dans leur profondeur. Le chémosis est considérable enveloppant pour ainsi dire une grande partie de la cornée qui, à cette période, ne paraît pas être changée. Le surlendemain tout rentre dans l'ordre. Le 3^e jour un changement apparaît sur la cornée qui devient plus nette le 4^e jour. La cornée s'éclaircit, les vaisseaux qui la sillonnent diminuent de calibre et deviennent filiformes. Vers le 4^e ou 5^e jour apparaît sur la conjonctive la formation du tissu fibreux déjà décrite. Vers le 7^e ou 8^e jour les petits vaisseaux qu'on n'apercevait qu'à l'éclairage oblique ne se voient plus. Vers cette époque, s'il existe (ce qui est fort probable) de l'opacité cornéenne, on assiste à l'éclaircissement constamment progressif de la cornée. Vers le 11^e ou 12^e

jour, c'est à dire après la 2^e injection, on constate un fait des plus curieux et des plus intéressants, que nous avons constamment vu chez nos malades: les gros vaisseaux du pannus, qui sont devenus filiformes mais qui restent encore visibles à cette période, présentent sur le limbe comme un arrêt brusque, une zone exangüe de 1 à 1½ mm. A ce niveau leur trajet donne tout à fait l'aspect d'une embolie, alors que les vaisseaux correspondants sur la conjonctive se continuent avec un volume environ 2 fois plus grand. Ce n'est que du 15^e au 18^e jour, c'est à dire après la 3^e injection, que les derniers restes des vaisseaux sanguins deviennent invisibles. De l'opacité cornéenne totale, il ne reste qu'un léger néphélion central qui diminue d'étendue petit à petit pour disparaître complètement.

Nous avons été très frappés durant le cours du traitement de notre premier malade Obs. I de voir l'état des conjonctives palpébrales, surtout au niveau des culs de sac, s'améliorer d'une façon très rapide et très marquée. (Trois à quatre jours après l'injection on constate l'affaïssement des follicules et des granulations conjonctivales. Mais la conjonctive parfaitement lisse reste hypérémiee. A la place des grosses granulations on voit de petites taches plus ou moins transparentes jaune gélatineux, grosses comme des grains de semoule. Une semaine ou deux de nitrate d'argent rend à la conjonctive son aspect et sa coloration normale.) Nous avons d'abord pensé que le cyanure avait agi sur les paupières de la même façon qu'il avait agi sur la cornée, par réaction, ou par formation de tissu cicatriciel ou par action bactéricide—Mais devant l'amélioration très nette et très rapide nous nous demandons si le cyanure n'aurait pas une action directe nous n'osons pas dire spécifique sur le virus trachomateux. Le nombre limité de nos expériences ne nous permet pas de nous prononcer. Il faudrait aussi pour cela des cas vierges traités exclusivement au cyanure. La date du Congrès ne nous a pas permis de le faire. Mais doré et déjà nous pouvons justifier de l'efficacité de la méthode par ces résultats.

La méthode est applicable à tous les cas et tous en bénéficient = *Restitutio ad intégrum* dans les cas légers de pannus (5 ou 8 jours après l'injection.)—Même résultat sur la conjonctive palpébrale dans tous les cas de la 1^e et 2^e périodes: toutes les granulations disparaissent en moins de 3 semaines, et la conjonctive récupère son aspect normal avec une ou deux semaines de nitrate d'argent ou de sulfate de cuivre—Dans les cas plus marqués de pannus, avec trouble considérable de la cornée, la guérison est moins rapide. On peut

espérer la disparition totale et complète des néphéliions pas trop anciens et du trouble diffus de la cornée. Le cyanure est sans action appréciable sur les leucomes étendues ainsi que sur l'hypertrophie du tarse et de la conjonctive, avec formation de tissu scléreux.

Dans les cas graves et anciens, combinés au nitrate d'argent ou au sulfate de cuivre, le cyanure diminue d'une façon considérable la durée du traitement. Dans tous les cas, le résultat fonctionnel est, comme on le voit, des plus encourageant.

Nous possédons vingt observations suivies plusieurs mois. Les résultats obtenus sont à peu près les mêmes dans tous les cas. Nous donnons seulement le résumé de quatre observations les plus caractéristiques.

OBSERVATION I.—Trachome très ancien non soigné 72 ans.

ODG.—Sécrétion larmoient. Blépharospasme, faux ptosis, épaississement de la conjonctive et du tarse. Pannus crassus, cornées ternes et très vascularisées empêchant de voir l'iris.

OD.—Mou et douloureux peut être iridocyclite. La vision de cet oeil est réduite à la perception lumineuse. VOG = compte les doigts à $\frac{1}{2}$ mètre. Du 19 Déc. 1920 au 16 Janvier 1921 traité sans résultat appréciable au sulfate de cuivre.

2 Janvier. Injection de cyanure à 1' O.D. Réaction assez marquée hématome sous conjonctival. Le 10 l'hématome est résorbé. Cornée moins terne et moins vascularisée. Conjonctive moins rouge considérablement améliorée. L'oeil droit propre et bien ouvert. Sulfate de cuivre.

Le 23 Janvier. Injection de cyanure à 1' O.G. Dix jours après le malade se conduit seul.

VOG. = $\frac{1}{16}$ Cornée presque complètement transparente, permet de voir une cataracte intumescence.

OD.—Infiltration légère et diffuse de toute la cornée cataracte en évolution.

Sort de l'hôpital appelé chez lui d'urgence. Collyre au sulfate de cuivre. Admis à nouveau le 11 Mars pour être opéré de cataracte. 12 Mars: Iridectomie préparatoire.

Un mois après extraction. Suites normales.

OBSERVATION II.—L'intérêt de cette observation est dans la guérison très rapide du pannus et des granulations par le cyanure; elle démontre bien la supériorité de ce procédé sur les moyens ordinaires.

M..... Jeune Grec, docteur en médecine. Soigné depuis 2 ans

chez lui et à Paris pour trachome par du nitrate d'argent et du sulfate de cuivre.

OD: Cornée normale. Quelques granulations typiques dans le cul de sac conjonctivale sup. et sur le bord du tarse.

OG: Photophobie et larmolement intenses. La conjonctive palpébrale très hyperémiée est entièrement couverte de granulations. Pannus typique du tiers supérieur. On devine, à travers le trouble de la cornée, la pupille en myosis. VOG = Voit passer la main.

10 Mars 1921. Injection de cyanure à 1' O.G. Réaction très intense. Le 15, Vascularisation occupant les $\frac{2}{3}$ sup. de la cornée.

Le 18, les vaisseaux diminuent pour disparaître complètement le 22. Nitrate d'argent en application quotidienne.

10 Juin. Cornée transparente, conjonctive parfaitement lisse sans granulation ni cicatrice, de couleur et d'aspect normal. VOG = 1.

OD: Soigné seulement par le nitrate d'argent présente une conjonctive légèrement injectée mais beaucoup moins qu'avant avec quelques granulations vers l'angle externe. VOD = 1.

OBSERVATION III AND IV.—Ces cas ne sont pas moins intéressants. Il s'agit de deux jeunes Syriens, refusés à 2 reprises par les Médecins inspecteurs du port de New York entre Août et Décembre 1920. Renvoyés à Paris pour la deuxième fois ils consultent à l'Hôtel-Dieu et subissent deux injections de cyanure dans les deux yeux suivies de deux semaines de sulfate de cuivre. Au mois de Mars 1921 c'est à dire deux mois après ils entrent à New York sans difficulté.

DISCUSSION

DR. J. M. PÉNICHET (Havana, Cuba): I have no doubt that Dr. Gemblath has been successful in the treatment of trachoma and its complications with the subconjunctival injections of cyanid of mercury. We all have had cases of trachoma radically cured by one of the many methods employed to treat this affection and, vice versa, we all have employed almost every known method on some difficult case without any or with very little success.

The point to consider is whether we are dealing with a simple case or with a complicated one. As a rule, trachomatous ulcers of the cornea and pannus do not yield to all forms of subconjunctival injections. That is why the results obtained by Dr. Gemblath should be taken into consideration.

To prove my statement I will say that four years ago I tried the subconjunctival autovaccine treatment introduced by Dr. Demaria of the Argentine Republic, and with only a slight modification in the technic I used it in twenty cases of simple trachoma and five cases of ulcers and pannus. My results were not as satisfactory as those of Dr. Demaria, and I must explain that I followed every one of these cases to the end of the series. My impression is that the organisms of trachoma seem to lose their virulence in the presence of or-

ganisms of ulcer and of pannus. In the five complicated cases better results were obtained.

The main objections to the use of subconjunctival injections of cyanid of mercury are the pain, the chemosis, and the psychologic influence of a bad appearance upon the patient and the family. According to Dr. Gemblath, aspirin given before, during and after the injections takes away all possibility of pain, and the bandage does away with the chemosis and swelling. In my experience I have found, nevertheless, that a large number of these patients will look for a less painful and easier method.

DR. JOSEPH KRIMSKY (Brooklyn, N. Y.): In Palestine, where I had charge of a number of schools and thousands of children suffering from trachoma, I began to use cyanid of mercury by massage instead of the bichlorid, using a 1 : 500 solution with vigorous massage. I had such good results that I conceived the idea of using it by subconjunctival injection, and I can testify to what Dr. Gemblath has said, that, in cases where there is an unpleasant swelling of the eyelids and chemosis it is not painful, and will subside in a week. Those cases in which I used the subconjunctival injection recovered in less time than those in which I had not used it. Then I tried it in more complicated cases where there was a thickened pannus, but I had no beneficial results and had to resort to operative treatment.

PROF. F. DE LAPERSONNE (Paris, France): J'ai constaté à l'Hôtel-Dieu de Paris, les résultats obtenus par M. Gemblath dans le traitement du trachome par les injections sous-conjonctivales de cyanure Hg. Ils sont extrêmement encourageants et ce traitement mérite d'être généralisé. Avec la technique employée, ce traitement est peu douloureux, malgré la tuméfaction considérable des paupières.

Je ne puis pas affirmer que dans ce cas le cyanure agit spécialement comme antiseptique; je crois plutôt que c'est par la réaction inflammatoire violente qu'il produit et par. L'apport d'élément fibrineux dans les tissus sous muqueux. Il agit un peu à la manière du jéquirity, mais plus profondément.

SURGEON JOHN MCMULLEN (U. S. Public Health Service): I am interested in the paper of Dr. Gemblath in so far as it would seem to afford us a remedy or a means of curing trachoma without resort to operative measures. I have not used this particular method, although, like all of us, have covered the ground pretty well in medical treatment, using practically all the remedies that have been recommended. I shall carry out the treatment in the Government Hospitals for the treatment of trachoma in the hope that we may relieve many of these cases. We have discarded medical treatment to a great extent except in so far as it is post-operative. Our treatment of trachoma is now altogether surgical.

DR. S. GEMBLATH (closing): La remarque de M. Pénichet, est très juste. La douleur causée par les injections nous a fait hésiter un moment, mais par l'usage de l'aspirine, nous avons pu la réduire à une simple gêne oculaire assez bien supportée.

Un mot pour répondre à Dr. Howe. Comme dans tous les cas traités nous

n'avons eu que des améliorations, sans accidents, nous n'avons pas hésité à employer le cyanure dans les cas même très légers et sans lésions cornéennes. Mais c'est surtout dans les cas de pannus et d'ulcères cornéens que ce procédé donne les meilleurs résultats.

Les injections de cyanure contrairement aux procédés chirurgicaux, sont applicables à tous les cas et par leur diffusion, leur action porte sur tous les points, atteints difficilement par les autres moyens. De plus, ils ne donnent pas naissance, comme le traitement chirurgical, à des cicatrices et à leurs conséquences fâcheuses.

HEREDITARY OCULAR DEGENERATIONS— “OPHTHALMIC ABIOTROPHIES”

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Most writers on hereditary affections of the eyes have omitted to draw any sharp line of differentiation between those in which the tissue involved has from the first been imperfectly developed, and those in which, after full development, degeneration sets in. As examples of hereditary maldevelopments may be mentioned such obvious structural imperfections as microphthalmia, ectopia lentis, and congenital cataract. Also conditions known to us best as functional disturbances, though doubtless having some anatomic basis, such as congenital color-blindness and night-blindness. The hereditary ocular degenerations of which the fullest information has been collected include the following affections: post-natal cataract; ocular palsies; retinitis pigmentosa; symmetric macular pigmentary degenerations; amaurotic family idiocy; Leber's optic atrophy; Doyne's "family choroiditis"; nodular and lattice-like degeneration of the cornea.

Widely different as these affections may at first appear, they will be found to present similar characteristics, which allows of them being grouped together in a classification of eye diseases founded on a pathologic basis. All these diseases are hereditary, in the sense that they may be met with in several siblings of the same generation, and all, with the exception of symmetric macular pigmentary degeneration and amaurotic family idiocy, in several different generations.

The lesions in all these diseases are bilateral, and in none of them in which pathologic investigations have been made do the findings suggest the presence of inflammation, but are such as indicate degeneration in certain definite groups of cells—cells which, having reached a full de-

gree of development and functional efficiency, have then undergone degeneration. The time of life at which the degeneration commences varies, as also does, what sometimes appears to be, the incidental exciting cause. The occurrence of the degenerations in several successive generations of the same family renders it impossible to attribute them entirely to the absorption of any exogenous toxic substance, or to the absence of any exogenous essential nutrient material, such as a vitamin.

So far as our knowledge at present goes there is no endogenous substance, such as the secretion of one of the endocrine glands, upon which the structures involved in these diseases are dependent for their vitality; nor do we as yet know of any toxic material developed endogenously, either by bacteria or as the outcome of some faulty metabolic process, capable of poisoning and slaying the cells picked out in these diseases. Even if such a toxin, or internal secretion, be discovered to account for the hereditary nature of the affections, it will still be necessary to assume some innate weakness in the cells attacked; or, in the case of some internal nutrient secretion, some innate tendency to a failure in its supply.

Sir William Gowers accounted for the hereditary character of certain degenerative affections of the nervous system, muscles, and epidermal appendages, presenting similar characteristics to the eye affections dealt with in this paper, to want of vital force in the structures involved, so that they are unable to maintain their nutrition beyond full development and, therefore, gradually fail and degenerate. They fail, as he says, "from imperfect life, from abiosis, in what may be designated abiotic atrophy, or abiotrophy."

He points out that, "besides general life, the termination of which involves that of every part of the body, many of these parts have their own vitality. Some of them slowly die, while the life of all the rest goes on without impairment. They die from many causes—some early, inevitably, from a very grave defect of vital endurance; some much later, the failure being but slightly premature; and some at various times, apparently from various causes. When the failure is early, it is often due purely to a defect in vitality, a defect which seems to be inherent, the tendency thereto inborn. We do not, indeed, apply the word 'death' to this slow decay of the elements; we speak of it as 'degeneration,' but the process is in many cases, perhaps in most, an essential failure of vitality, and I think it is instructive to consider the degeneration in this aspect."

The term vital force, in the way in which it is here used, may be defined as "a force the possession of which differentiates living from dead matter." Though we know little as to its real nature, it is convenient to have a name to apply to it, just as in algebra we use a symbol to represent an unknown quantity. This vital force, as we know it, exists only in connection with cell protoplasm, and manifests itself by what we term irritability. For its maintenance, the cells possessing it require the capacity of taking up certain substances from the fluid media surrounding them, and of discharging other waste products—a process probably capable of a physicochemical explanation.

In fission fungi and many unicellular protozoa the vital force may apparently be indefinitely prolonged, for these simply formed organisms multiply by division, so becoming transformed into two new individuals without leaving any corpse behind.

In a germ cell of multicellular organisms, as the result of fertilization, the vital force, instead of becoming gradually exhausted and leading to degeneration and death of the cell, regains fresh vigor, causing it to multiply, develop, and evolve into a new being.

In the cells of multicellular organisms, which have become much differentiated from germ cells, and have acquired new and highly specialized functions, the vital force is always in a state of transition. These cells are always either in a state of evolution to maturity or in a state of involution to decay. At times the process is so slow that it might almost be thought that no change was taking place, but slow though it be, and minute the alterations that occur, it surely and steadily goes on. "For so we ripe and ripe and then we rot and rot."

The time required for the vital force to complete the cycle of changes which take place from birth to death varies considerably in different species of animals, in different individuals of the same species, and physiologically in the several tissues of which an animal is composed.

Among mammals the vital force is of much longer duration in elephants and man than in horses and dogs. Among birds it is of much longer duration in parrots and geese than in fowls and thrushes. In the tortoise it is of longer duration than in any other vertebrata, one such animal is estimated to have lived for two hundred years.

Among human beings longevity is frequently found to be a family characteristic. Here are three examples: Mrs. Amelia Spurgeon, the aunt of the great preacher, recently passed her one hundred and second birthday. Her mother lived to ninety, and her brother and two

sisters all passed the eighties. In a recent copy of a daily paper there was an account of an octogenarian, a Mr. Gibbins, who had eight brothers and sisters, not one of whom died before reaching the age of eighty.

Thomas Parr, who was born in Shropshire in 1483, is reported to have reached the age of one hundred and fifty-two, and was buried in Westminster Abbey. The celebrated Harvey examined his body after death and was unable to discover any organic disease. Parr did not marry until the age of eighty-eight; he had a son who lived until the age of one hundred and twenty-seven.

Physiologically, in the human body, the vital force fails sooner in some tissues than in others, *e. g.*, the thymus gland is highly developed during fetal life, reaches its maximum size during the third year of life, and shows signs of degeneration which rapidly progress after the age of ten. Other early physiologic failures of vital force, with which most of us are unfortunately only too familiar, are touchingly alluded to in the following verse from W. S. Gilbert's comic opera of "Patience":

Silvered is the raven hair,
 Spreading is the parting straight,
 Mottled the complexion fair,
 Halting is the youthful gait:
 Hollow is the laughter free,
 Spectacled the limpid eye,
 Little will be left of me,
 In the coming by-and by.

As Sir James Paget³ wrote in his famous Lectures on Pathology: "It is natural to become feeble and infirm, to wither and shrivel, to have dry, dusky, wrinkled skins, and greasy, brittle bones, to have weak, fatty hearts, blackened, inelastic lungs, and dusky thin stomachs, and to have every function of life discharged feebly, and as it were wearily; and then, with powers gradually decreasing, to come to a time when all the functions of bodily life ceasing to be discharged, death, without pain or distress, ensues." He goes on to say that such a death is rare, and that he has only seen two or three such cases.

In other words, our bodies are not constructed like "The Deacon's Masterpiece; or the Wonderful One-Hoss Shay," described by Oliver Wendell Holmes:

"That was built in such a logical way,
 It ran a hundred years to a day."

and then:

"went to pieces all at once,—
All at once and nothing first,—
Just as the bubbles do when they burst."

To again quote Sir James Paget; he says: "The changes of natural degeneration in advanced life have a direct importance in all pathology, because they may guide us to the interpretation of many similar anomalies which, while they occur in earlier life, we are apt to call diseases, but which are only premature degenerations, and are to be considered, therefore, as methods of atrophy—as defects, rather than as perversions, of the nutritive process—or as diseases only in consideration of the time of their occurrence." This was written in 1853, and it is an interesting anticipation of Sir William Gowers' theory of "Abiotrophy," or degeneration of tissues due to defective vitality.

Metchnikoff⁴ would have us believe that the symptoms we speak of as senile degeneration are not a natural sequence of life, ushering in its close, but the outcome of auto-intoxication. In his writings, however, he frequently speaks of "the cycle of life" and of "natural death," which latter he defines as a "phenomenon that is intrinsic in the nature of an organism and not the mere result of an external accident." What apparently he does not recognize is that the cycle of life may be of variable duration in the different tissues of an individual, and the intrinsic phenomenon, "natural death," may take place sooner in some cells of the body than in others. Doubtless some tissues in which the cycle is drawing to a close are likely to succumb to some toxic influence earlier than they otherwise would, in which case the change produced might be attributed entirely to the toxin, the weakened resistance power of the tissue having been overlooked.

It is as unreasonable to attribute all variations in the time of degeneration of tissues to toxic influences as it would be to account for all variations in the time of development of tissues to such influences. Just as we sometimes meet with precociousness in development, so sometimes we meet with precociousness in degeneration. It is with some of the precocious degenerations of the tissues of the eye that I now proceed to deal.

HEREDITARY POST-NATAL CATARACT OR ABIOTIC CATARACT

Under the heading of cutaneous abiotrophy Sir William Gowers¹ described the early baldness met with in the males of the family in

several successive generations, the essential cause of which is the failure of the life of the hair-follicles of the scalp. He also spoke of early grayness of the hair as a qualitative failure, an enduring defect of one function of the follicles.

Another very common form of degeneration met with in a structure derived from cuticular epiblast is opacity of the lens of the eye, the outcome of a premature failure in the vitality of its fibers, a condition which may, I suggest, be aptly termed abiotic cataract.

That these changes are due to defective vitality is shown by their inherent character. Nettleship⁵ states that: "Post-natal or acquired cataract is often hereditary, and quite a number of pedigrees have been collected by many observers."

Such post-natal cataracts have been described according to age of onset as "senile," "presenile," and "juvenile." But, as Nettleship says: "This subdivision is, no doubt, arbitrary, and does not correspond with any differences in the cause or character of the cataract, but is of some practical convenience."

He collected and analyzed a large number of pedigrees of post-natal cataract, and, commenting on the age of onset, remarked: "In one and the same family hereditary cataract often begins at about the same age in all who have it. But exceptions to this are very numerous for, as we have just seen, hereditary cataract often occurs at an earlier age in the children than in the parents, whilst in those of the same generation it frequently begins at about the same age in each."

In 1917 I operated for cataract at the Moorfields Hospital on two members of a family of seven. The five other members of the family and their father had previously been operated on for cataract at the same hospital. These facts I was able to verify by reference to the in-patient notes. I was further told that my patient's two paternal uncles had been operated on for cataract, one at St. Thomas' Hospital and the other at Cardiff. Of the family of seven two were males and five females. Their ages at the time they were operated on were as follows: forty-two, fifty-six, forty-nine, sixty-three, fifty-six, fifty-eight. The age of their father at the time of operation was sixty.

In a family such as this, in which so large a number of its members became affected with early senile cataract, it seems necessary to assume some inherent defect in the vitality of the lens-fibers which results in their degeneration.

To speak of certain forms of post-natal cataract as abiotic does more than simply describe them as due to degeneration. I propose to show

that it enables us to understand their pathology more clearly than has previously been possible.

In considering the degenerative processes which occur in the structure of the lens it is first necessary to recognize that there is a physiologic degeneration constantly taking place in its fibers which does not result in the formation of opacity, and which, in an anteroposterior section of an adult lens, can be seen in all its stages in passing from the periphery to the nucleus.

Newly formed lens-fibers, such as are seen close to the capsule, form long flat bands with regular margins, rounded or hexagonal in transverse section, and containing rounded or elliptic well-staining nuclei. Older fibers, further from the capsule, become denser and flatter; their nuclei show at first an aggregation of chromatin toward the periphery, and later disappear altogether, clear oval spaces being left at the sites they occupied, which, in still older fibers, also disappear. The oldest fibers of all, those in the center of the lens, are harder and more condensed. They have crenated margins, the projections and indentations of which fit tightly into one another, and they are entirely devoid of any trace of nuclei. This gradual hardening of the lens-fibers is spoken of as sclerosis.

In pathologic degeneration of the lens, such as is met with in senile cataracts, the first change observed is the formation of clefts between the fibers in its peripheral parts. In these clefts the interfibrillar fluid accumulates, and in hardened sections of the lens presents the appearance of irregularly shaped, coagulated masses, or of spheroidal bodies, the so-called Morgagnian globules. The formation of these clefts is usually attributed to an excess of the physiologic shrinking of the nucleus of the lens. In the adult lens, and to a larger extent in the senile lens, the nucleus is quite hard, due to the physiologic shrinking and degeneration which have taken place in its fibers. It seems difficult to understand how there can be any increase in the physiologic shrinking of this already horny, sclerosed part of the lens. The region in which the clefts are formed and where the opacity begins is where the lens-fibers have not, in the normal state, undergone much shrinking or degeneration; and regarding the condition as due to abiotrophy, it would be natural to assume that the clefts are due to a premature shrinking and degeneration of what should be well-nourished fibers, rather than changes in a part of the lens where metabolic processes are practically at a standstill. Though the lens-fibers bounding the clefts at first show but little change beyond some condensation of their sub-

stance, such as is met in the physiologic degeneration process, later on their substance becomes granular, fatty globules may form in them, and they break down into the spaces formed between them by their shrinkage, and thus the cataractous process progresses.

HEREDITARY OCULAR PALSIES. ABIOTROPHY OF THE MUSCLES OF THE EYELIDS AND EYEBALLS

In discussing the various forms of hereditary paresis of the muscles of the eyes and eyelids it will be well first to quote some of Gowers'¹ remarks on muscular abiotrophy in general. In his first lecture on the subject he wrote as follows:

"In the various forms of idiopathic muscular atrophy, in which there is a primary atrophy of the muscular fibers, we have examples of a true abiosis. To all these primary myopathies it has become customary to apply the term 'muscular dystrophy,' and the custom is convenient, if not quite accurate. The term thus includes both simple muscular atrophy and its well-known congener, pseudohypertrophic paralysis. In these the muscle-fibers, after full development, cease to maintain their nutrition. They slowly waste, and a large number—most of them in many parts, all in some—perish. The connective tissue between them overgrows. Its increase may fail to maintain the normal bulk of the muscles, so that they waste conspicuously, sometimes extremely. In other cases this tissue-weed, as we may regard it, presents a more luxuriant development, and produces fat-bearing cells which so much increase the bulk of the muscle as to cause the enlargement of pseudohypertrophy."

In another lecture² on the same subject he wrote:

"The nutrition of the muscle depends on that of the nerves through which its function is called forth. If the nerves slowly degenerate, so does the muscle; if rapidly, from descending irritation, the muscles undergo speedy complete degeneration. Yet the muscle has a life which we may call organic, belonging to it as a structural entity, in consequence of which it may undergo morbid changes, apart from the nervous system, and may fail to live on, though the nerves preserve an unimpaired vitality. This failure is what I have called muscular abiotrophy—failure of nutrition from defective vitality—and for brevity we call it myopathy."

Among the muscular abiotrophies which he thus defines he includes the "facio-scapulo-humeral type," also designated the "type of Landouzy-Déjérine." In this affection there is extreme wasting of

the muscles picked out, including the orbicularis palpebrarum. No matter how long the duration of the malady, it seems to remain purely muscular, the nervous system, even the motor nerves, remaining unchanged. It is a family and hereditary disease, and may be transmitted by either male or female parent; it affects both boys and girls alike.

In 1915 I showed⁶ a girl, aged seven, who had bilateral facial paralysis. At that time there was no affection of the muscles of the shoulder-girdle, though there was some paralysis of the extensor muscles of the leg, causing her to walk with a peculiar gait, her toes turning inward. She had the typical expressionless, so-called myopathic, face. She was unable to close her eyes from paralysis of the orbicularis muscles, and her lower lids fell a little away from the eyeballs, causing epiphora. There were no symptoms pointing to any involvement of the nervous system, and the case appeared to be one of purely muscular atrophy.

Fuchs⁷ in 1890 reported five cases in which bilateral ptosis was the only symptom of disease. In two of them, aged thirty and sixty respectively, the ptosis appeared early in life, was hereditary, and in course of time became complete. The other three, aged forty, sixty, and fifty-six years respectively, developed ptosis late in life; it slowly increased and became almost complete. There was no history of syphilis in these cases, and no evidence of brain disease. The upper eyelids were thinned to such an extent that the anterior parts of the eyeball showed plainly through them. There was also shrinking of the orbital fat in the immediate vicinity of the levator, as shown by falling in of the lid just below the orbital rim. Fuchs removed and examined a small portion of the muscle in one of his cases, and found the fibers much thinner than those from a healthy muscle; the nuclei were considerably increased in number, and there was marked pigmentary degeneration inside the sarcolemma but no fatty degeneration was found. In some parts the fibers themselves appeared normal in structure, the connective tissue between them being, however, increased in amount. Fuchs was of opinion that his cases were best explained by a primary atrophy confined to the levator palpebræ muscles, an affection which, up to that time, had not been described.

In 1909 I published the notes⁸ of what I took to be a similar case to those described by Fuchs of bilateral ptosis, unaccompanied by paralysis of any other muscles. Like all Fuchs' cases, the patient was a female, and like some of his cases, there was a slight amount of ptosis,

dating either from birth or from early infancy. That the ptosis had steadily increased was well shown by the examination of a series of photographs she had had taken at different ages. At the time she came under my observation she was sixty-nine years old. She also had a very unusual circumferential encroachment by the limbus of the conjunctiva on to the cornea in the two eyes. I cut away a strip of the levator palpebræ muscle in each eye, as Fuchs did, and found it very atrophic; unfortunately, I did not have it examined microscopically.

In 1900 W. M. Beaumont published⁹ the account of a family in which several members of four different generations suffered from ophthalmoplegia externa. The disease was never congenital, but always appeared in adult life; it was slowly progressive, and never appears to have had a fatal termination. The characteristics of the members of the family were to enjoy a long life and to have troops of children. The cases, most fully examined, showed complete paralysis of the levator palpebræ on each side, and very limited action of the recti muscles in any direction. In one case it was noted that the action of the orbicularis muscle was very feeble. In none of the cases were the intra-ocular muscles involved.

McMullen and Hine,¹⁰ in a recent paper on chronic progressive ophthalmoplegia externa, mention that: "Wilbrand and Saenger¹¹ grouped as a clinical entity certain cases of external ophthalmoplegia, which, in their opinion, had definite characteristics, which separated them not only from the congenital cases of this affection, but also from the cases definitely due to some toxic cause or to syphilis, or associated with wider spread manifestations of nervous disease." These cases, according to these authors, have the following characteristics: "There is a gradual onset, generally in infancy or early childhood, more rarely in later life, of a bilateral, progressive paralysis of the external eye muscles, not associated with other signs of disorder of the nervous system nor with fever. The disease may come to a standstill permanently, or for long periods, at any stage of its development, but generally ends in complete, or nearly complete, external ophthalmoplegia. In most cases both sides are affected from the beginning, but sometimes it appears on one side long before the other; often the two sides are unequally affected in the early stages. Ptosis is generally the first sign, and may be for a long time the only one. When the muscles of the eyeball become affected, diplopia may be complained of, but this symptom is only rarely noted, principally because the paraly-

sis develops gradually, and often symmetrically, on both sides, and because it frequently comes on in childhood before the function of binocular vision is fully developed. The progress is often extremely slow, and thirty or forty years may elapse between the appearance of the first symptoms and the development of complete ophthalmoplegia. The general health is not affected, and there is a noteworthy absence of signs indicating disease in other parts of the nervous system."

Wilbrand and Saenger¹¹ collected records of 32 cases of this affection. McMullen and Hine, in their article, refer to two others described by Ayres¹² and Altland,¹³ and give notes of three of their own. In Ayres' case the maternal grandfather of the patient was said to have been similarly affected. The following case, evidently belonging to the same class, has recently come under my observation:

Frederick R., aged forty-three, stated that his eyelids began to droop twenty-eight years previously, and that shortly afterward he was operated on for it by Mr. Lawford. His condition was improved, but the drooping had persisted. He came to me on account of some conjunctivitis in one of his eyes, and I then discovered that, in addition to bilateral ptosis of a very marked degree, he also had ophthalmoplegia externa. He had only very slight power of movement of either eye laterally or vertically, and practically no rotatory movement. His pupils were equal and active, and he had no more defect of accommodation than such as might be expected in a man of his age with a slight degree of hypermetropia. His occupation was a carman; he had never experienced any diplopia, or found any difficulty in driving about the crowded streets of London. He has six children—none of them or, so far as he knows, any other member of his family has suffered from an affection of the eyes similar to his. He is a strong, healthy man, shows no sign of any affection of his nervous system. His Wassermann reaction was tested and found negative.

In discussing these cases it is first necessary to draw a distinction between them and cases of congenital defect in the movements of the eyeballs and eyelids, a class of cases dealt with very fully by G. Heuck¹⁴ in 1879, and by Lawford¹⁵ in 1888. The former found, by post-mortem examinations, that in these congenital cases the defect of movement was due either to absence of the muscle, its imperfect development, its insufficient length, or its faulty attachment to the eyeball. In the class of cases here under consideration there has for a time been good movement of the eyelids and eyeballs, and then gradual and progressive failure has set in. In the one there is from

the first a defect of development, in the other degeneration sets in after full development.

Most writers on these hereditary ocular palsies, and on the cases presenting similar symptoms which arise sporadically, regard them as due to lesions in the nuclei of the nerves supplying the affected muscles. It is, however, possible that the primary condition may be a degeneration of the muscle-fibers and that the nerves supplying them may be unaffected. Whichever be the true explanation, seeing that the affection is in some instances a hereditary one, and that it is not due to syphilis, it may appropriately be regarded as of an abiotic nature—either an abiotrophy of the cells of the nerve nuclei, or an abiotrophy of the muscle-fibers.

The assistance which might be obtained by examination of the electric reactions in these cases to determine the nature of the muscular weakness is not available. The exact primary nature can certainly be decided only by postmortem examination, but up to the present no such examinations have been carried out.

The order in which the different series of paralytic affections of the muscles of the eyelids and eyeball are referred to above has been arranged with a purpose. The first-mentioned were cases of the facio-scapulo-humeral type, in which there was inability to close the eyes from paralysis of the orbicularis palpebrarum muscle; and then a case of bilateral facial paralysis without any affection of the muscles of the shoulder-girdle. These cases are undoubtedly of a myopathic nature. Next, Fuchs' cases of bilateral ptosis were quoted, some of which were hereditary; these he regarded as due to primary atrophy of the muscles, a view which received support from the appearance of a strip of one of the affected muscles examined microscopically. Following on these Beaumont's family, with ophthalmoplegia in four different generations, was described. In one member of the family, in addition to the ophthalmoplegia, there was some paresis of the orbicularis palpebrarum muscle. Finally came the group of cases separated off as a clinical entity by Wilbrand and Saenger, in which there was chronic progressive bilateral paralysis of all the orbital striated muscles, the unstriated intra-ocular muscles invariably remaining unaffected. In this group the muscles affected are never confined to those supplied from one nerve nucleus, and the paresis of some of the muscles often remains for a long time incomplete. Many of the cases in this group have been watched for prolonged periods without developing any symptoms due to disturbances of the nervous system.

These facts taken together offer, I suggest, strong circumstantial evidence in favor of the view that cases of bilateral ophthalmoplegia, like cases of bilateral facial paralysis and cases of bilateral ptosis, are due to a primary degeneration in the muscle-fibers.

“RETINITIS PIGMENTOSA” OR ABIOTROPHY OF THE RETINAL NEURO-EPITHELIUM

In a paper published in 1919¹⁶ I collected together a considerable amount of evidence to show that the affection commonly known as retinitis pigmentosa should really be regarded as a primary degeneration, or abiotrophy, of the neuro-epithelium of the retina. It will be here only necessary to summarize the evidence set out in that paper.

Nettleship,¹⁷ in 1907, made a most comprehensive collection of all the data concerning retinitis pigmentosa then available, and described the disease as “the result of a tissue liability present, though seldom manifest, at birth, and known to be so often hereditary that we may be sure it is so also in many cases where the proof is wanting.”

Wagenmann¹⁸ showed, by division of one of the posterior ciliary arteries in animal eyes, that the nutrition of the outer layers of the retina depend on the choroidal circulation. In the area supplied by the divided vessel both choroid and retina atrophied, the latter becoming adherent to the former and also pigmented.

Frequently after sclerosis of the choroidal vessels, due either to syphilis or to senility, pigmentation of the retina is observed. From this, and from Wagenmann's experiments, it had been inferred that the pigmentation of the retina in retinitis pigmentosa is also due to sclerosis of the choroidal vessels. Pathologic examinations of eyes in cases of retinitis pigmentosa, at different stages of the disease, and made by different observers, have shown it may occur without any such thickening of the choroidal vessels being present.

W. T. Lister,¹⁹ in 1903, examined the eyes of a man who died at the age of sixty, who, at the age of thirty-eight, had been found by Nettleship to have advanced retinitis pigmentosa. Lister found that though the choriocapillaris was atrophied, the choroidal vessels seen in section showed no thickening of their walls. The layer of rods and cones was altogether absent, its place being taken by laminated fibrous tissue.

Stock,²⁰ in 1908, examined microscopically an eye with typical retinitis pigmentosa, excised during life for an adjacent carcinoma, and fixed immediately in Zenker's fluid. He found a practically normal choroid. The primary changes appeared to be a degenera-

tion of the bacillary layer of the retina, followed by degeneration of its nervous elements and disturbance of the pigment epithelium.

Ginsberg,²¹ in 1908, examined histologically the eyes of a man known to have had retinitis pigmentosa for ten years, who died of tuberculosis at the age of thirty-five years. The vascular system of the choroids in these eyes showed no changes. The outer layers of the retinae were the parts most affected, the neuro-epithelium being almost entirely destroyed. He suggested that its degeneration was due to a congenital weakness in the standard of functional activity imparted to these cells, so that they had not grown in proportion to the demands made upon them and therefore gradually disintegrated; *i. e.*, to abiotrophy.

Suganama,²² in 1912, examined microscopically the eye of a man, aged sixty-seven, with typical retinitis pigmentosa, removed on account of a hypopyon ulcer. There was no sclerosis of the choroidal vessels and the choriocapillaris was normal, except in a few areas where it was absent or imperfect. There was complete absence of the rods and cones. The retinal nervous elements were atrophic, and the neuroglia had proliferated.

The evidence afforded by these pathologic investigations seems conclusively to show that retinitis pigmentosa may arise and exist for many years unaccompanied by any thickening of the choroidal vessels. Further, that the disease starts in the retinal neuro-epithelium, which, having attained its full normal development, then degenerates. As it is a hereditary condition, this tendency to degenerate must be due to some inherent weakness in the affected cells.

Atrophy of the percipient elements in a sensory organ is necessarily followed by atrophy of the nervous elements in connection with them, and a decrease in the vascular supply to the part. So in the retina in retinitis pigmentosa, following on the atrophy of the rods and cones, there is atrophy of the ganglion-cells and other nervous elements in the retina, together with a dwindling of the smaller blood-vessels of both the retinal and choroidal circulations.

As often happens, when the highly specialized elements of a structure degenerate, the less highly organized supporting framework of the tissue tends to increase. It will be well for me to quote here what Gowers¹ wrote of this matter in connection with neuronie abiotrophy of the central nervous system, because it applies so well to the retina in retinitis pigmentosa. He says: "Whenever the nerve elements waste there is always an overgrowth of the interstitial neuroglia, the

connecting and supporting tissue which lies between them. This overgrowth may be, indeed, on first inspection, the most conspicuous element, and its aspect has led the progress to be termed "sclerosis." I say "its aspect" because there is generally no increase of consistence in the part so changed. This is generally diminished; the interstitial tissue, which looks so fibrous and firm under the microscope, is really softer than the nerve elements that have perished.

"The two elements, the neural and the neuroglial, seem to have a common but inverse vitality; it is in consequence of this that the interstitial tissue overgrows when the nerve elements decay. The overgrowth seems to coincide with the very commencement of the decay, and may be at first the most conspicuous.

"It is especially luxuriant where the connective tissue is naturally most abundant—about the vessels and at the surface of the spinal cord beneath the pia mater."

Having described the pathologic findings in this disease I will now show how well they help us to explain the sequence of the symptoms met with in connection with it.

The first symptom a patient complains of in retinitis pigmentosa is night-blindness; this precedes the appearance of any pigmentation. As Nettleship¹⁷ pointed out, the term "retinitis pigmentosa sine pigmento" "usually signifies nothing more than the initial stage of the ordinary disease." The part of the retina first involved is the perimacular zone; a ring scotoma is found to precede any contraction of the periphery of the field. Pigmentation of the retina when it makes its appearance is also first seen in an intermediate zone between the macula and the periphery. It is not until the later stages of the affection that the retinal blood-vessels become narrowed and the optic disc acquires its characteristic waxy appearance.

In the dark-adapted eye the part of the retina which is most sensitive to light is that surrounding the macula. Experiments have shown that its sensibility increases centrifugally with each degree until 10° to 20° from the fovea, where the maximum is reached. On the other side of this maximum the sensibility decreases toward the periphery in approximately concentric circles. Degeneration of the percipient elements of the retina, in the part most acutely sensitive to light, accounts, therefore, for the early occurrence of the night-blindness.

In a typical case of retinitis pigmentosa the inner border of the ring scotoma corresponds, as a rule, to about the 15° or 10° in perimeter

charts. That is to say, the part of the retina in which failure of function commences is that in which its neuro-epithelium first attains its full development. The macular area, together with the nerve-fibers proceeding from it, is both ontogenetically and phylogenetically of late development.

Pigmentation of the retina is due to the migration of pigment epithelial cells into its substance. The reason why it is such a characteristic feature of the disease we term retinitis pigmentosa is that the disease commences in the rods and cones; the gaps left in the *membrana limitans externa* by their disappearance form tracks through which the pigment cells can make their way. We know that these pigment epithelial cells physiologically possess the power of ameboid movement. When stimulated by light, they throw out filamentous processes between the outer segments of the rods and cones, and on removal of the stimulus retract these processes into the body of the cell. They possess a capacity for positive phototactic movement which is kept in check only by their anatomic relations; when these are altered by the atrophy of the rods and cones, together with the nerve elements of the retina, so that only a network of neuroglial tissue remains, then the pigment epithelial cells, attracted forward by the stimulus of light, are able to make their way into the innermost layers of the retina, where they accumulate in the lymph-spaces around the blood-vessels and give rise to the characteristic branching patches.

The waxy appearance of the optic disc in retinitis pigmentosa, which is different from that met with in any other affection, is attributable to an overgrowth of its neuroglia. In rare cases hyaline bodies, sometimes in grape-like clusters, are seen protruding from the surface of the disc. In microscopic appearance and in their reaction to chemical reagents these hyaline bodies are found to resemble those so commonly met with on the inner surface of the elastic lamina of the choroid. These latter are due to some perverted activity of the pigment cells—*i. e.*, cells which are derived from the outer layer of the secondary optic vesicle. Cells having a similar embryonic origin are found in connection with supporting neuroglia of the optic nerve. It would seem probable, therefore, that in retinitis pigmentosa, where there is an overgrowth of neuroglia in the optic nerve, the hyaline nodules met with on its surface are due to some perverted activity of the neuroglia cells.

Retinitis pigmentosa is not infrequently associated with other forms of hereditary degenerative conditions, the commonest of these

being deafness and idiocy. Nettleship¹⁷ estimated that 33 per cent. of persons with retinitis pigmentosa may be deaf, and at least 4 per cent. of deaf-mutes may have retinitis pigmentosa. The deafness in these cases is always bilateral and is never recovered from. Even when accompanied by dumbness it may have been acquired, according to Politzer, as late as the age of seven years. The pathologic changes found in the labyrinth of deaf-mutes are comparable to those found in the retina in retinitis pigmentosa, and may be summarized as degeneration of the neuro-epithelium, atrophy of the nerve-fibers, and a new formation of fibrous tissue. It is then highly probable that, as in retinitis pigmentosa, the affection being primarily a degeneration of the neuro-epithelium, which is hereditary, it should be classed as an abiotrophy.

SYMMETRIC FAMILIAL PIGMENTARY MACULAR DEGENERATION.
PRIMARY DEGENERATION, OR ABIOTROPHY, OF THE CONES AT
THE MACULA

De Wecker, in 1868, divided cases of retinitis pigmentosa in two groups: One which occurs in children, with comparatively rapid loss of central vision, and which is frequently associated with mental defects; the other, comprising the ordinary cases, beginning with night-blindness early in life, progressing very slowly, the central vision remaining unaffected until late in the disease, and in connection with which mental failure is but rarely met.

Nettleship¹⁷ also described atypical cases of retinitis pigmentosa with central changes. He pointed out that the pigmentation in them was in the form of scattered dots, instead of bone-corpuscle-shaped patches; and that the patients, instead of being night-blind, preferred a dull light.

Several cases have been recorded of recent years of primary macular pigmentary degeneration, occurring in several members of the same family, often in association with cerebral degeneration, but sometimes without. These cases have been described under the following headings: "Family Cerebral Degeneration with Macular Changes" (Batten and Mayou); "Maculocerebral Degeneration (Familial)" (Oatman); "Progressive Familial Macular Degeneration" (Darier). None of these titles is satisfactory. From the two first it would be inferred that the cerebral changes were an essential part of the disease, and from the third that the disease tended to progress to complete loss of sight. I and others have recorded cases which have manifested

nomenal symptoms, some of them having been kept under observation for several years. The disease, when it commences at the macula, generally remains confined to that region and does not progress beyond it.

The reason that the pigment patches in retinitis pigmentosa assume a bone-corpuscle shape is due to the pigment epithelial cells making their way into the lymphatic sheaths around the retinal blood-vessels; as there are no blood-vessels in the retina at the macula, when the pigment epithelial cells migrate into the retinal tissue in that region they do not form branching patches, but rounded dots.

The histologic changes in an eye of a typical case with symmetric pigmentary changes at the macula in each eye have been investigated by Mayou.²³ He found that the sclera and choroid were practically normal, the pathologic changes being confined to the retina. Briefly, they consisted of a complete disappearance of the cones and the neural elements of the retina, together with some increase of the supporting neuroglia and migration of pigment epithelial cells, to a slight extent, into the outermost layers of the retina. Mayou, in commenting on this case, inclined to the view that the primary change was in the ganglion-cells of the retina, and that the disappearance of the cones and the migration of pigment were secondary. Coats,²⁴ however, in commenting on this case in a later article, wrote very shrewdly as follows: "There is, I believe, no known instance of a disease which first attacks the inner retinal neural elements and then the outer, although instances of the opposite sequence of events might be cited; nor does destruction of the inner layers from whatever cause produce any consecutive secondary change in the outer, however long the duration of obstruction of the central artery or primary optic atrophy, in which the rods and cones and outer nuclear layer remain permanently intact."

When the general nervous system is affected in connection with this pigmentary degeneration, there is a progressive dementia and paralysis ending in death. After some years of normal development the mental degeneration sets in, accompanied sometimes by epileptic fits; before death the patients become noisy, dirty in habits, and develop a spastic condition of the limbs. In two cases examined post-mortem by Dr. F. E. Batten²⁵ diffuse degenerative changes were visible microscopically, affecting the ganglion-cells in the cerebrum, cerebellum, and spinal cord. These changes, he says, were similar to those found in amaurotic family idiocy.

From the account already given of retinitis pigmentosa, and from

that which follows of amaurotic family idiocy, it will be seen that this primary pigmentary macular degeneration presents some resemblances to each of those affections, and also some striking differences. These resemblances and differences are set out in tabular form as follows:

	Retinitis Pigmentosa	Macular Pigmentary Degeneration	Amaurotic Family Idiocy
Age and progress	Commences early in life and progresses slowly.	May commence at age of six or not until the second decade; rapid failure of sight and then stationary.	Onset at age of three to six months; rapid failure of sight; death in two years.
Heredity	More than one generation affected; no racial proclivity.	Familial, no racial proclivity.	Familial; only met with in the Jewish race.
Visual defect	Night-blindness followed by annular scotoma; central vision last affected.	Central scotoma; no contraction of field; no night-blindness.	Central loss of sight early, progressing to complete blindness.
Condition of retina	Primary affection of bacillary layer at equator; later formation of branching patches of pigmentation.	Primary affection of bacillary layer at macula; later formation of dots of pigmentation.	Primary affection of ganglion-cells, causing white opacity around macula.
General nervous system	Sometimes mental weakness, cause unknown.	Sometimes dementia; ganglion-cells affected as in amaurotic family idiocy.	Always dementia. All ganglion-nerve cells in body involved.

The eye symptoms in all three affections are due to some innate tendency to degeneration in the cells of the retina. The difference which they present in the three affections is due to the difference in the cells involved. Any one of them may be accompanied by a similar innate tendency to degeneration in ganglion-cells of the central nervous system. All three affections may, I suggest, be conveniently classified as varieties of abiotrophy.

FAMILY AMAUROTIC IDIOCY (TAY-SACHS' DISEASE). ABIOTROPHY OF THE GANGLION-CELLS OF THE RETINA

A considerable amount of evidence has accumulated of recent years as to the pathologic changes met with in the nervous system and retina in cases of family amaurotic idiocy.

All writers on the subject are agreed that there is no evidence of inflammatory changes in the affected parts, and that the disease is due to a primary change in the cells themselves. Gordon Holmes showed that this change was not due to an arrest of their development or to any bacterial toxin. The essential histologic features of the disease are: a progressive loss of the Nissl substance in all the neurons of the body, and the increase of the neuroglia fibril substance to an abnormal degree. A chemical examination of the brain in two cases by Sidney A. Mann showed: (1) A decrease of nucleoproteid, which may be associated with the disappearance of the Nissl substance in the neurons; and (2) The increase of simple proteid, which may be correlated with the increase of glial fibrils. Both Gordon Holmes²⁵ and Sir Frederick Mott²⁶ attribute the cell changes to some faulty biochemical process in the protoplasm of the cells. The latter sums up his conclusions on the matter as follows:

"The fact that it affects the children of Jewish parents suggests that it owes its origin to some racial inborn tendency to neuronie decay, probably associated with some exciting or predisposing factor connected with an altered condition in the chemical composition of the blood, whereby the normal biochemical interaction of the nucleus on the cytoplasm and the environmental lymph on the neuron is interfered with. 'A cell nourishes itself and is not nourished' is as true for the highly complex and specially differentiated nerve cell, with its multiple processes and their arborizations, as for a simple unicellular organism. The nucleus is the trophic center of the nerve-cell, and possesses the specific inherent energy upon which the cell depends for its vital activities and durability. We may, therefore, suppose that this extraordinary neuronie regressive metamorphosis is brought about by a conspiracy of morbid factors, viz., an inherent racial lack of specific neuronie energy and some general alteration in the chemical composition of the blood, either by the existence in it of a neurotoxin or the failure of some chemical substance in sufficient quantity for the building up of the nucleoproteid substance of the nervous system."

The cause of the opacity of the retina, seen around the macula, in these cases was for some time a matter of uncertainty, due to the difficulty in obtaining specimens free from postmortem changes and suitably fixed. Within two hours of death changes in the retina set in, causing it to swell, become rucked, and then present microscopically the appearances of edema or of a hole at the fovea. This edematous change was the most striking feature which I discovered in the first

specimen of the sort which was examined microscopically, and I attributed the opacity seen ophthalmoscopically to edema. Since then fresher material has been obtained, fixed in Zenker's solution, sections of which have shown a complete absence of any edema, but well-marked changes in the ganglion-cell and nerve-fiber layers. There can now be no doubt that it is the altered condition of the ganglion-cells which give rise to the opacity. Where they are most numerous around the macula, there the opacity is densest. The changes in the ganglion-cells of the retina resemble those found in the ganglion-cells of the nervous system elsewhere. At first they appear somewhat swollen, then there is a gradual progressive loss of Nissl substance, followed by the formation of vacuoles in the cytoplasm, and finally shrinkage or disappearance of the cell. As Coats pointed out, the long continuance of the opacity of the retina around the macula may be correlated with the long time the degenerative change in the cells is going on before they finally disappear. Very different is this to the short duration of the opacity of the retina, similarly situated, in cases of embolism of the central artery of the retina, which is due to a coagulative necrosis of the ganglion-cells, after which they become rapidly absorbed and disappear.

Transverse sections of the optic nerve in advanced cases of amaurotic idiocy show that the nerve-fibers have nearly all disappeared, the few remaining fibers being fine ones and irregular with varicosities.

There is some difference of opinion as to the appropriateness of the use of the term abiotrophy as descriptive of the changes met with in family amblyopic idiocy. Sachs²⁷ says he gladly accepts it as indicating the nature of the changes met with. Gordon Holmes,²⁵ on the other hand, writes: "The cell changes have not the characters of a simple atrophy; in fact, they seem to be due to an excessive growth of the protoplasm which later undergoes degenerative changes. This fact is not in favor of Sachs' hypothesis that the pathology of the disease can be described by the term abiotrophy (a term suggested by Gowers to represent an inherent defective vitality of the cell), or of Schaffer's suggestion that it may be explained by Edinger's 'Ersatz-theorie,' which assumes that the elements which are inherently feeble undergo degeneration when exposed to the strain of life to which they are not normally resistant."

There can be no doubt that the change in the ganglion-cell is a form of hereditary ocular degeneration. The degeneration, as already mentioned, is of a different character to, and of slower progress than,

that which takes place when these cells are deprived suddenly of their nutrient supply by occlusion of the central retinal artery. If, as Mott suggests, the degeneration is in part due to the failure in the inherent specific trophic influence of the nucleus of a nerve-cell, upon which the vitality of the whole neuron depends, then the term abiotrophy is as appropriate as any that has been invented under which to classify the affection. Even though the first stage in the trophic disturbance presents the appearance of hypertrophy, the final stage in the process is invariably the premature death and atrophy of the affected cell.

HEREDITARY OPTIC ATROPHY. "OPTIC ABIOTROPHY"

The occurrence of what was termed "amaurosis" in several members of the same family was first described by Beer in 1817. Leber²³ published his classic paper on hereditary optic atrophy in 1871. Many cases of the same description have since been recorded, and much has been written on the subject, but, so far as I am aware, no pathologic examination has been made of these cases and we are, therefore, still ignorant as to the real nature of the disease.

Gowers,²⁹ in 1904, described the affection under the heading of "Optic Abiotrophy," and spoke of it as follows:

"Since some cases were discerned by Leber, much attention has been given to the form of optic atrophy which occurs in families, sometimes through more than one generation, soon after adult life is attained. Similar cases occur in sporadic form, isolated, as do other family maladies. The facts suggest that the inherent vital energy of these structures is inadequate to maintain their nutrition much beyond full development, so that they gradually fail and degenerate. They fail from imperfect life, from abiosis, in what may be designated abiotic atrophy, or abiotrophy. The same atrophic failure is met with in other parts of the nervous system, as in Friedreich's disease, and conspicuously in the muscles in the varieties of muscular dystrophy.

"It is noteworthy that even vital failure is often associated with extraneous influences. Even in the cases in which the family disposition is most marked, the onset is often the immediate sequel of some adventitious cause. It follows some acute specific disease, or, in males, excessive smoking, and this in cases in which the family tendency is so marked as to compel us to regard the excitant as merely such, an opinion which is confirmed by the slow progress of the atrophy after the cessation of the immediate influence. In such cases the interstitial

tissue undergoes overgrowth, in consequence of the solidarity of the vital tendency of the two structures, both of which arise from the same embryonal elements. When the vitality of the higher, neural elements fails, that of the residual, neuroglial elements becomes exuberant, and may even display an energy which carries it beyond the strict limits of the neural failure. Thus we are able to understand some features of this form which at first seem mysterious. It must be remembered, also, that the defect in vital endurance varies in degree and in extent even within the range of the tissue which presents it. How grave it may be we cannot know until it is revealed by time, but the influence of a powerful excitant may at least afford ground for hope, and indicate room for treatment. It should be remembered also that, in such maladies, therapeutic measures, when most effective, may only prevent further failure."

In 1916 J. H. Fisher,³⁰ without taking into consideration Gowers' views on the nature of this affection, put forward a new hypothesis as to its causation; he wrote as follows:

"It has occurred to me that if a disturbance of the pituitary body of temporary duration and moderate degree can be imagined, such a lesion might be adequate to explain the phenomena of Leber's hereditary optic atrophy, in which case the inherited tendency would not lie in a special vulnerability of the macular fibers of the optic nerves, but in a liability of the pituitary body to such limited disorder as I suggest. A priori it is to me much easier to imagine an inherited tendency to disorder of the hypophysis than a family tendency on the part of the papillomacular fibers of the optic nerve to degenerative or inflammatory attacks."

The points upon which Fisher based this hypothesis are:

I. That cases of bilateral temporal hemianopsia, due to pressure on the chiasma, have been known to develop, in the first instance, a central scotoma.

II. That individuals who develop hereditary optic atrophy are often of what Leber described as the neuropathic type. They are subject to frontal headache, vertigo, and epileptiform attacks; they also experience subjective phenomena of light and color. Fisher suggests that these symptoms may be taken as suggestive of pathologic disturbance or excessive physiologic activity on the part of the pituitary gland.

III. That the affection often coincides in its onset with puberty, or in women, the climacteric period, at which times, Fisher suggests,

variations in the size of the pituitary body are most likely to be met with.

IV. That glycosuria is sometimes associated with a loss of central vision, similar in some respects to that met with in Leber's disease, and that disorders of the posterior lobe and pars intermedia of the pituitary body is frequently accompanied by glycosuria.

V. That lesions of the optic chiasma due to pituitary body enlargement or growth may, in the early stages of the affection, give rise to a very mild papillitis, and that this symptom has also been noticed in the early stages of Leber's disease. While in the later stages of both affections signs of optic atrophy present themselves in the optic disc.

VI. That in some cases of the disease which have been examined with the *x*-rays an abnormal appearance has been observed in the vicinity of the sella turcica.

Difficulties present themselves in the acceptance of either Gowers' or Fisher's theory as to the pathology of hereditary optic atrophy.

If we regard the disease as a form of abiotrophy, it would be necessary to determine the structure in which the degeneration commences. As the failure of sight in the most typical cases is restricted to the central region, it would be most natural to locate it in the ganglion-cells from which the fibers start, which constitute the papillomacular bundle. In diseases in which the ganglion-cells of the retina are known to be affected with degenerative changes, such as embolism of the central artery of the retina, quinin amblyopia, and amaurotic family idiocy, for a time, varying induration in the different affections, an opacity of the retina is seen ophthalmoscopically. No such opacity has been observed in Leber's disease, though there is often, at first, a temporary appearance suggesting edema about the optic disc. That temporary opacity of the retina is not an essential preliminary phenomenon in the onset of primary atrophy of the optic nerve we know, from its absence in connection with primary atrophy of tabes. In this latter affection we have no certain knowledge as to which part of the affected neuron is primarily involved.

One of the most constant characteristics of diseases attributable to abiotrophy is their incurability; there is a degeneration due to loss of vitality, and once lost, it is not restored. There are cases of hereditary optic atrophy on record in which complete or partial recovery has taken place; thus Nettleship writes:

"I find records of at least 25 affected persons (22 males, 3 females)

in 16 genealogies who recovered either perfect or quite useful central vision; minor degrees of improvement are probably rather common."

"In the same genealogy, and even in the same sibship, some may recover and others not." "A very important feature in these cases is the length of time that may elapse before notable improvement of sight begins,—often twelve or eighteen months, and in one case, if we can believe the history, as much as three years."

Gowers would probably attribute recovery, or partial recovery, in such cases to variations of intensity in "the adventitious cause," which, he pointed out, in the quotation given above, is necessary, in addition to the family disposition, for the production of this disease.

The most tangible factor for consideration in connection with Fisher's hypothesis is the appearances shown by *x*-ray examination.

The table on p. 128 gives the result of examination of the sella turcica by *x*-rays in Fisher's,^{30, 31} Pollock's,³² and Dr. James Taylor's³³ cases, together with three cases I have had under my own observation, one hereditary and two sporadic.

The results of *x*-ray examinations, which are given in the table, seem to show that there may be some association between the loss of central vision met with in Leber's disease and changes in the vicinity of the sella turcica. Fisher naturally regards this as affording support to his hypothesis, that the affection is due to changes in the pituitary body. Indeed, it was with the idea of testing his hypothesis that he first undertook these *x*-ray examinations.

So far no well-recognized case of hereditary optic atrophy has been known to develop bitemporal hemianopsia, and we do not know of any hereditary form of pituitary disease, whereas we are well acquainted with bilateral degenerative conditions affecting the nervous system, such as Friedreich's disease, due to atrophy of the motor neurons, a typical abiotrophy. It would seem well, therefore, to consider how Gowers' theory of abiotrophy, applied to Leber's optic atrophy, might account for changes in the vicinity of the sella turcica, such as are revealed by *x*-ray examination. Gowers, as quoted above, speaks of overgrowth of neuroglia in the optic nerve in Leber's disease; this is not the result of direct observation, as the optic nerves in this affection have not yet been examined pathologically. He must have inferred the presence of such overgrowth from the presence of similar overgrowth in the nervous system in other forms of abiotrophy. There is, however, ophthalmoscopic evidence which affords support to the view that, associated with atrophy of the nerve elements in this disease,

Recorder	Sex	Age	Duration of Affection	Results of x-ray Examination
Fisher	Male	14	2 yrs.	Sella turcica shows no departure from normal.
Fisher	Female	11½	6 mos.	Sella turcica not enlarged, outline not distorted, but the depression filled in with something which gives a cellular or honey-comb-like shadow.
Fisher	Male	22	3 mos.	Sella turcica outline slightly indistinct posteriorly, but it is not enlarged or distorted.
Fisher Sporadic case	Female	13½	5 mos.	Pituitary fossa showed nothing abnormal. Seven months later fossa was found to be normal in size and outline, but there was a domed shadow which formed a roof over the sella turcica. Four months later still sella turcica again showed domed shadow roofing completely the fossa.
Fisher Sporadic case	Female	49	1 wk.	Doubtful enlargement of the pituitary fossa and some want of distinction of the posterior clinoid processes. A second examination, made nine months later, showed more marked approximation of the anterior and posterior clinoid processes.
Pollock	Female	11	6 mos.	Situated a little below the center of the sella turcica, a shadow like a small bean, with the concavity downward.
Pollock	Male	8	1 mo.	Same appearances as in the case last recorded, that of his sister.
James Taylor . .	Male	52	A few months	Sella turcica long and shallow; clinoid processes certainly not normal.
James Taylor . .	Male	60	10 yrs.	Sella turcica shallow, and clinoid processes reduced in size.
Treacher Collins	Male	42	6 mos.	Sella turcica distinctly large, and the opening into it apparently wider than usual.
Treacher Collins Sporadic case	Male	24	6 wks.	The floor of the pituitary fossa is very shallow and flattened, also irregular. The anterior clinoid processes are unduly pronounced, and the posterior processes barely evident at all.
Treacher Collins Sporadic case	Female	33	14 mos.	No abnormal changes seen in the region of the sella turcica.

there takes place an excessive formation of neuroglia. After the disease has been in existence for some time pallor of the outer half of the disc commences, and spreads over its whole surface. Leber himself described this pallor as due to increase of the connective-tissue elements of the nerve. If, then, there is an overgrowth of neuroglia at the head of the nerve in this disease, we may be sure that there is also a similar overgrowth along the track of the papillomacular fibers elsewhere. The greatest thickening of this neuroglia would be expected to occur where the affected fibers in the two nerves came together, at the chiasma. A thickening of the chiasma, due to overgrowth of neuroglia, might press down the pituitary body, just as an enlargement of the latter may press up the chiasma; or might also, in a skiagram of the sella turcica, give rise to the appearance of a roof-like covering to it, or of a bean-shaped body overlying it.

All these speculations as to the real nature of hereditary optic atrophy might be cleared up by the pathologic examination of a typical case. The best way to make known the urgent need of pathologic evidence of this description is, I think, to proclaim it to the members of a large congress such as this.

DOYNE'S "FAMILY CHOROIDITIS," OR ABIOTROPHY OF THE RETINAL PIGMENT EPITHELIUM

R. W. Doyme³⁴ described a form of family choroiditis in 1899 and 1910. In his last communication concerning it he summarizes the condition as follows: "It first appears in early adult life, but much more commonly later. It may either affect the disc neighborhood or the macula neighborhood, or the disc macula area. It consists of circular patches of exudation; these increase during middle age, and at least set up some irritation and pigmentary disturbance, for, though pigment is not always present, in some cases there is a good deal to be seen. During this stage the sight, though affected, is not grossly interfered with. In old age the condition passes into atrophy, with a corresponding degree of failure of sight."

He had met with the condition in two families, affecting several members of each, in the same and in different generations. A similar condition in the members of another family, a mother and two daughters, was described by Major Mould³⁵ in 1910.

At the conclusion of his communication in 1910 Doyme wrote: "I am keeping my attention carefully on some of the older cases, and

I hope, if I live long enough, I may be able to bring before the Society some microscopic sections." Doyne's health failed him in 1913, so that he had to retire from practice, but that year one of the elderly patients with this affection, whom he had been keeping under observation, died, and he obtained the backs of his eyes, which he handed over to me for pathologic examination. In making the examination I had no preconceived notion or theory as to what the condition was and wrote the following description of the histologic appearances which the specimen presented. Mr. Doyne showed the specimen at the Oxford Ophthalmological Congress in 1913, and the description of it was published in that year.³⁶

"In sections through the posterior part of the eyeball in the region of the macula and optic disc are seen the following changes. Situated between the retina and choroid is a new formation of a hyaline substance. It commences on each side of the optic disc close to its margin, and extends inward from it about a distance of two discs' breadth, and outward a distance of six discs' breadth. Its external surface has a regular contour following natural curve of the choroid. Its inner, or retinal, surface presents several rounded nodular elevations with depressions between them. The thickness of the hyaline substance varies, therefore, considerably in different parts. It shows in places very definite lamination, and for the most part is entirely free from cells, although here and there a cell with a flattened nucleus is seen embedded in it.

"The choroid external to the hyaline tissue shows marked thinning, and in places complete absence, of its internal capillary layer. The vessels of the outer layer appear, however, abnormally large, but their walls are not thickened. In the choroid, on the outer side of the optic disc, there is a small area of round-cell exudation, but apart from this there are no signs of inflammatory disturbance. Where the hyaline substance is present, the elastic lamina of the choroid or membrane of Bruch cannot be satisfactorily differentiated. At the periphery of the hyaline substance, on each side, the pigment epithelium can be seen extending for a short distance over its inner surface as a single layer of cells. Large parts of the inner surface of the hyaline substance are, however, devoid of any lining of pigment epithelium, while here and there irregular collections of pigment epithelial cells are met with. The outer surface of the retina has become much disorganized by the formation of the hyaline tissue. The rod-and-cone layer has become entirely destroyed where it is situated, also to

a very large extent the outer nuclear layer. In some localities the granular layers are thickened, the fibers crossing them being stretched out with spaces between them. The inner layers of the retina show but little change."

To this description of the appearances of the sections, I, in 1913, added the following remarks:

"The hyaline substance in these sections presents the same histologic appearances and staining reactions as the nodules of hyaline commonly met with in a similar situation in many degenerative conditions of the choroid. Such nodules are often termed 'Drusen.' The hyaline continuous substance in this specimen is peculiar in forming such a long layer. In other conditions it is usually in the form of isolated nodules. The ophthalmoscopic changes, known as 'Tay's choroiditis,' are generally regarded as being due to such nodules. Many different views have been put forward to account for the formation of these hyaline nodules. They have the same characteristics as the elastic lamina of the choroid, and it seems probable that they are, like it, the product of the pigment epithelial cells. Some of the hyaline tissue in this specimen looks very like a number of superimposed layers of the elastic lamina. Further, it seems likely that the primary change was in the pigment epithelium, and that the changes in the choroid and retina around are secondary to the pressure caused by the formation of the hyaline substance."

From the above description of pathologic appearances and remarks it will be seen that, in this family affection first described by Doyne, we have to do with a primary degeneration of the cells derived from the outer layer of the secondary optic vesicle, and not with an inflammatory affection of the choroid. The white appearance seen in the fundus ophthalmoscopically is not due to an inflammatory exudation, but to the formation of hyaline substance by the cells involved, which is, on their part, a degenerative process. A degeneration of this nature handed down from one generation to another, and affecting several members of the same family, may, I suggest, be aptly included in the class of affections described by Gowers as "abiotrophies."

The affection is, I think, one more commonly met with than the scant attention it so far has received in ophthalmic literature might lead one to expect. The slow progress of the affection, and the absence of symptoms in its early stages, render its family nature likely to be overlooked. As pointed out by Gowers, though abiotrophies are usually hereditary conditions, sporadic cases of the same nature some-

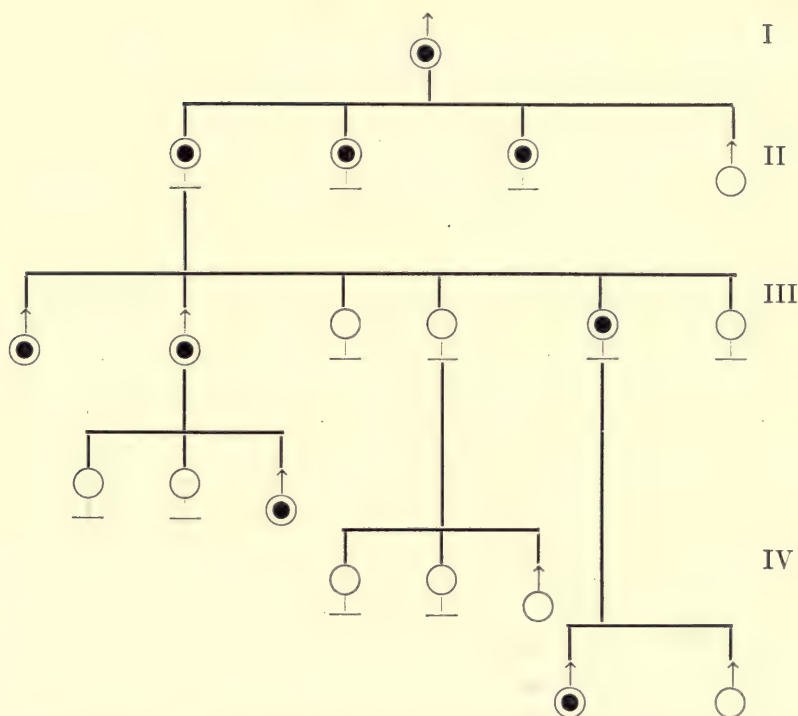
times occur, so it is in this hyaline degeneration of the pigment epithelium of the retina. This year I have seen two cases in which the condition was well marked, though no history of failure of sight in other members of their families could be obtained. From what Doyne has said it is evident that the absence of a history of heredity is not sufficient evidence to exclude the presence of the disease, as it may be present in other members of the family though unsuspected for many years.

HEREDITARY DEGENERATION OF THE CORNEA. ABIOTROPHY OF THE CORNEAL NERVES

A bilateral affection of the cornea, starting usually about puberty, and manifesting itself by the formation of opaque nodules in the superficial layers, was first described by Groenouw³⁷ in 1898, under the name "knötchenförmige Hornhaut-Trübungen." His description of the appearances in his two cases remains substantially true for the many others which have since been recorded. He says: "The disease consists in the development of numerous small, rounded or irregular, gray, discrete opacities in the otherwise clear cornea. The larger opacities attain to a diameter of nearly a $\frac{1}{4}$ mm., and between these lie much smaller, dust-like gray points; they, for the most part, are situated in the central region of the cornea, and rather avoid the marginal zone. The larger spots slightly raise the epithelium and thus give a certain minute irregularity to the surface. The opacities appear by degrees without any inflammatory reaction, and may remain for years unchanged."

The hereditary nature of the affection does not seem at first to have been noted. In 1902 Marcus Gunn³⁸ recorded four cases in a family of ten. Spicer,³⁹ in 1904, described a family in which three generations were affected. The following is the pedigree of a family, some of the members of which I have had under my own observation, in which four generations were affected.

In none of the cases recorded has there been any evidence to show that the affection was of a syphilitic nature. Several observers have described the histologic appearances of small pieces of the cornea, either scraped off the surface or trephined. Paderstein had the opportunity of examining the whole eye in a case, it having been removed after death from a patient who died from suppurative meningitis following an injury to the head.



The changes found are situated in the basal cells of the epithelium, between the epithelium and Bowman's membrane, in Bowman's membrane itself, and in the superficial layers of the parenchymatous tissue of the cornea. Paderstein⁴⁰ said that, on first looking at the specimens, one gets the impression that the disease affects primarily Bowman's membrane; closer inspection, however, shows that the primary seat of the disease is in the basal epithelium. He considers that, through a degenerative process taking place in the epithelial cells, the nucleus is destroyed and the protoplasm changed into a hyaline substance. This hyaline substance tends to accumulate on the surface of Bowman's membrane. Fuchs,⁴¹ in his most recent article on the subject, states that: "The main points are that in this affection two primary changes are present, *i. e.*, the deposition of a substance that is often in layers immediately under the epithelium, and gives an acidophile reaction, and a second basophile, granular substance in the cornea proper." He is inclined to think that the acidophile is a later product than the basophile substance, since the latter has been found

in all the reported cases, while the former has not been always present. He describes as accessory changes the alteration in the epithelium, the thinning and destruction of Bowman's membrane, and the separation, swelling, or destruction of the corneal lamellæ. Fuchs considers the affection to be of the nature of a dystrophy, and comparable to other similar processes found in old age, as the result of disturbance of general nutrition or, as a result of interference with the internal secretion of certain glands, as in myxedema.

Besides the nodular opacities above described, hereditary degeneration of the cornea may take on the appearance of a trelliswork, or lattice-like opacity, confined, as in the nodular variety, to the central part of the cornea and to its anterior layers. The hereditary nature of the affection was first pointed out by Haab,⁴² who met with it in a lad aged sixteen, and also in his maternal aunt and uncle. Dimmer recorded three cases in two brothers and a sister. Freund⁴³ described 15 cases in two families—seven in one and eight in the other, in four generations.

That the two forms of hereditary degeneration of the cornea, the nodular and the lattice-like, are really only different manifestations of the same affection, was demonstrated by Doyne and Stephenson,⁴⁴ who wrote as follows: "The two diseases, then, agree in several particulars—as, for example, that they begin at about the period of puberty; that they are accompanied by insignificant signs of inflammation; that they are slowly progressive; that they probably represent not an inflammatory but a degenerative process. Then, lastly, there is the fact that the sensitiveness of the cornea, particularly as regards the central region, may be impaired; and, finally, that both conditions tend to run in families." They record seven cases of bilateral central degeneration of the cornea in three generations of a family; in some of these cases the disease belonged to the lattice-like and in others to the nodular variety.

It will be seen, then, that these degenerative, bilateral diseases of the cornea belong to the same class of ocular affections as those already described in this paper, and correspond with those degenerations met with in other parts of the body, attributed by Gowers to premature loss of vital force, or abiotrophy. The pathologic investigations which have been made, though enabling us to classify the disease as a degeneration, do not supply us with sufficiently definite evidence to determine in which structure in the cornea the degeneration primarily starts. Two points in the clinical history of these affections suggest

that possibly the nerves of the cornea might be the primary seat of the disease. Fuchs, in describing his nodular cases, spoke of the superficial sensitiveness of the cornea being lessened. Jacqueau described a family in which eight cases of lattice-like variety were met with, and in whom the sensibility of the cornea to touch was diminished.

It would seem most probable that an opacity occurring in a number of different cases in the form of a meshwork, the lines composing which, by focal illumination, look like glass thread (Haab), or birch-twig-like branches radiating from the periphery toward the center (Freund), must have some anatomic basis. It is clear, from the description of the cases, that these lines do not represent tracks left by blood-vessels. In no stage of the disease does the cornea become vascularized, and its peripheral parts always remain clear and free from lines. Any tracks left by blood-vessels would necessarily extend out to the margin of the cornea, to join the vessels from which they were originally derived. The only other anatomic structure we know of in the cornea presenting the appearance of branching lines is its nerve-fibers.

The most recent investigations into the arrangement of the nerve-fibers in the cornea are those of Dogiel,⁴⁵ who adopted a method of staining them with methylene-blue. According to him, two-thirds of the nerve-trunks which enter the cornea pass into the anterior lamellæ, where they form what is termed the primary or fundamental plexus; the remaining third are distributed to the posterior layers. The nerves, from which the central portion of the fundamental plexus is derived, enter the cornea at a deeper level than those which constitute its peripheral parts. The fundamental plexus is composed of a network of fibers in the anterior layers of the stroma, where the fibers meet, forming nodal points at which nuclei are often to be seen; these are now regarded as belonging to the delicate investing sheaths of the nerves, and not, as formerly, to ganglion-cells. The spaces left between the fibers are traversed by interlacing, delicate fibrils. From the fundamental plexus fibers pass forward, through channels in Bowman's membrane, and on its outer surface form the subepithelial plexus. From the subepithelial plexus fibrillæ pass in between the epithelial cells, terminating either as round or pyriform end-bulbs or as terminal fibrillæ. The fibrillæ of the fundamental plexus, lying between the lamellæ of the cornea, come into close relation with the corneal corpuscles, and it has been a matter of much discussion as to whether or not there is any direct continuity between the cells and the fibrillæ.

A comparison of this description of the corneal nerves with the distribution of the opacities in the cases of hereditary degeneration of the cornea suggests much similarity of arrangement. The nerve-fibers form a plexus in the anterior layers of the substantia propria, a subepithelial plexus, and terminate in end-organs situated among the epithelial cells. In hereditary degeneration the pathologic changes are found in the same situations. The central part of the fundamental plexus and of the subepithelial plexus, which latter is constituted of fibers derived from the former, take origin from different nerve-trunks to those from which the peripheral parts of these plexuses proceed. Hereditary degeneration of the cornea is confined to the central part, and there also anesthesia is noted to have been most marked, *i. e.*, in a region which receives its nerve supply from one set of nerves. The fundamental plexus is composed of a network of fibers crossing and intersecting one another, similar in appearance to the arrangement of the lines of opacity met with in the lattice-like type of hereditary degeneration of the cornea.

In hereditary degenerations of nervous structures in other parts of the body, associated with the atrophy of the nervous tissue, there are always changes in the surrounding supporting fibrous tissue. If, as I suggest, this hereditary degeneration of the cornea is a primary dystrophy of its nerve-fibers and nerve end-organs, then we should expect to meet with thickening of the neuroglia tissue surrounding the nerves, some hyaline degeneration in it, and a formation of spaces filled with coagulum representing those previously filled by the nerve tissue. These changes resemble those which have been described as met with in cases of nodular degeneration of the cornea. It is possible that the different appearances in the arrangement of the opacity, which has been met with clinically in these diseases, may be accounted for by a difference in the part of the nervous system of the cornea which is primarily attacked. For instance, if it started in the fundamental plexus in the substantia propria, we should expect the opacity in the first instance to be of the lattice-like variety; whereas if it started in the end-organs in the epithelium, we should expect it to start in the form of opaque dots or nodules.

For any final decision as to the primary seat of this disease, further research is necessary. I would suggest that the employment of Dogiel's method of methylene-blue staining to a fragment of an affected cornea, removed either by scraping or by trephining, might be of considerable assistance in clearing up the problem.

TREATMENT

In dealing with the treatment of these various forms of hereditary ocular degeneration it is first well to point out that a medical man is called upon to certify that life is extinct, as well as to prescribe remedies for various ailments. Every medical student has to learn the signs of death in an individual, so it is well for an ophthalmic surgeon to learn to recognize the signs of death in a tissue of that organ of the body with which he specially deals.

The recognition that these maladies are due to premature death of the tissues involved does not offer much hope for their alleviation by therapeutic measures. It may, however, save us from trying to revivify dead matter by iodid of potassium, vapor baths, or other measures of that description, which only raise illusive hopes in our patients, cause needless expenditure of money on their part, and are doomed to failure.

Bichat defined life as "the sum of the forces which resist death," and though, like all other definitions of life which have been attempted, it leaves something to be desired, it is useful in connection with the subject now under consideration. One of the forces which resist death is inherent, and varies in its duration and degree. Though at some future date it may be accounted for by physicochemical changes, at present we know too little concerning it to attempt to control it. Other forces which resist death are connected with the environment of the organism or of its tissues. It is essential that it should have an adequate and suitable supply of nutrient material. If the vital force in a tissue is inherently weak, the adequate supply of suitable nutrient material may delay its final extinction; which, on the other hand, might be accelerated by the presence of toxic substances in the fluids in which the tissue is bathed. In these hereditary degenerations, to delay the failure of vital force by maintenance of nutrition, and to remove any source of toxemia, is all we can hope to effect by medicinal measures. In some of the affections dealt with operative procedures may alleviate some of the inconvenience caused by them. Thus abiotic cataracts can be removed and the sight restored. In bilateral ptosis, the removal of a strip of the tarsal plate will do away with much of the discomfort which drooping of the eyelid gives rise to. In cases of retinitis pigmentosa trephining operations, by producing a state of hypotony, have caused dilatation of the retinal vessels, and tended to delay the atrophic process. In some cases of nodular opacity of the cornea its anterior layers have been scraped away or excised by a trephine, with some slight improvement of sight.

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DISCUSSION

PROF. C. E. FINLAY (Havana, Cuba): In connection with Mr. Collins' interesting paper I want to state that in Cuba I have had opportunity of observing a good many of these cases. Congenital cataract is frequent; also retinitis pigmentosa, post-natal cataract, and the lattice-like degeneration. In most of these cases we have been able to see a certain amount of hereditary influence. That is especially marked in retinitis pigmentosa and post-natal cataract.

We have a province in Cuba which before we had railroads was completely isolated, and there we have had a number of intermarriages in the same family, cousins marrying cousins, uncles marrying nieces, etc., so that the number of cases of congenital cataract is greater there than in the rest of the island. In one family cataract was observed in seven different members. In this same province a Chinaman married a white woman, and they had six children, five of whom had congenital cataract. As regards the lattice-like and nodular opacities, I have been able to observe them in the same family. I remember a mother, three daughters, and a niece, all of whom had the nodular variety or the lattice-like opacities. In all of these cases it was limited to the female members of the family.

DR. F. H. VERHOEFF (Boston, Mass.): The term "abiotrophy," I take it, implies something inherent in the cell which causes it to die, irrespective of any external influence. It seems to me that the term has no advantage over the term with which we are more familiar, and that is "premature senility." The term "premature senility" would not necessarily mean immediate degeneration, and we know that senile changes do not always consist of death of the cells, but sometimes of hyperplasia. Mr. Collins' hypothesis is an attractive one as regards senile cataract. In cataracts definitely known to be due to disturbance of nutrition the opacities are different from those in senile cataracts. They begin in the posterior cortex whereas those of senile cataracts begin as peripheral striæ. A typical senile lens, however, is a sclerosed lens, hence a senile cataract does not represent premature senility of the lens as a whole. We do, however, occasionally see sclerosed lenses in younger people. I recently saw a man of fifty who had a sclerosed lens such as you would expect in a man of seventy or eighty.

In regard to Doyne's choroiditis, I understand Mr. Collins regards this as an exaggerated form of Tay's choroiditis. The changes in this are primarily proliferative in nature; they indicate not merely death of the cells, but that the pigment cells undergo proliferation. The hyaline material I think is produced by secretion of the cells, as Coats pointed out. So in this condition you see the term "premature senility" would be better than "abiotrophy."

In regard to the treatment of some of these conditions I should like to suggest that it would be well to try the x-ray. We know that the latter in weak doses will stimulate resting cells, and in stronger doses will destroy proliferative cells. About a year ago I began to employ it in retinitis pigmentosa. A sufficient time has not elapsed to tell whether or not it has been of real benefit, but it certainly has done no harm. The difficulty has been to decide upon the dosage and frequency of exposure.

DR. E. E. BLAAUW (Buffalo, N. Y.): Since the previous Congress we have made certain advances in our knowledge of the formation of cataract, and I think the term "abiotrophy" is an excellent one.

The first lenticular changes appear at the age of twenty. If we dilate the pupils, we will find spots in 20 per cent. of eyes, which will increase in extent and intensity and progress to senile cataract. The changes appear in the cortex, at the place where outer and middle third of the radius meet (the coronary cataract of Vogt). With the slit lamp also can be demonstrated convincingly that these earlier cortical opacities do not appear directly below the capsule but always at a certain distance away from it. As a rule, senile cataract does not begin in the posterior cortex.

I agree with Mr. Collins that in many of these cases we find heredity plays an important rôle.

DR. GEORGE F. LIBBY (Denver, Col.): We owe much to Edward Nettleship and his colleagues for putting the subject of ocular heredity on such a firm foundation; and I think it is a very happy circumstance that one of Nettleship's co-workers should bring before us this notable paper. Reading Mr. Collins' paper carefully in the pre-session volume I felt that at last we had a nomenclature that was satisfactory. I have not been fully satisfied with the rather hazy terms we have used in describing this condition.

In reading the conclusions of the paper I felt that a little more hope was held out in the matter of hereditary degeneration—perhaps more than the author has indicated in the summary of his paper just presented. At any rate, we get a definite idea as to what is actually hopeless, and what we may so represent to our patients. It seems to me that in the conditions which Mr. Collins has described we should look out particularly for the hygiene of such patients. I think they deserve a little more care, direction, counsel, and caution along hygienic lines than the majority of patients.

DR. EDWARD JACKSON (Denver, Col.): I feel that Mr. Collins has done us a real service in tracing the line of communication between the widely separated individual conditions that he has grouped, such as ptosis, ophthalmoplegia, Leber's disease, pigmentary degeneration of the retina, and others that we have thought of separately, not connected with each other. He certainly has given a new view with reference to these conditions, although the new view that we first get may be different from the final view.

There are two or three points that I think are illustrated in the discussion. One of them has just been referred to, and that is the hopelessness. It has always been assumed that death of tissue is a hopeless condition. It is hopeless after it has occurred, but the family tendency in that direction need not by any means be hopeless, and, as I think Mr. Collins mentioned, and as the last speaker has indicated, it is rather an incentive to study more closely the conditions which hasten or retard the death of the tissue in premature senility, or abiotrophy.

As to the connection between heredity and these affections, I can conceive that as the somatic death of the individual is hastened or retarded by certain influences, so the tendency to early death of the cell tissue, a degeneration that is not hereditary, may be so influenced. There may be a tendency to early

death of certain tissues of the eye, but that does not entirely remove retinitis pigmentosa from syphilitic disease. They are often confused, and the syphilis may have been the cause in one patient, of tendencies that are similar to the hereditary tendencies of another patient. In this connection I wish to refer to Leber's disease. It has been assumed very generally that it is a primary disease of the optic nerve in patients with a hereditary tendency. One case that I saw about thirty years ago (the only case I have ever seen in the beginning), and that I have heard from within a very few years, has gone through his life giving the ordinary history of hereditary optic atrophy. That patient entirely deceived me by lying about his family history, but one of my colleagues knew the family and quickly traced the history—he knew the man's ancestors had been similarly affected, so that with the subsequent course of the case I was able to place it in that connection. That case illustrated the fact that, even if we have not an enucleated eyeball to study with the microscope, we have the facilities to study the living eye with the ophthalmoscope. In that case the patient ran through a course of central retinitis with some small hemorrhages, not marked, confined entirely to the macula, and ending with an atrophy of the optic nerve corresponding to this central scotoma, with no visible change in the macula after the first few weeks or months. Now I can conceive, taking that for an illustration, that some temporary condition to which that family undoubtedly was extremely sensitive may have appeared in that young man to start a local process in the retina which gave the picture of Leber's atrophy.

So, while we have connected these different conditions with tissue death, and that is right, yet I think treatment is valuable. It does not render them hopeless, and it gives to us not only a good ground for prognosis, but leaves us a wide field for study, with the hope that in the future the family perhaps may benefit from our labors.

DR. MARY BUCHANAN (Philadelphia): About 1904, a Hebrew woman who had retinitis pigmentosa, brought her infant with amaurotic family idiocy to the Polyclinic Hospital. The child died of pneumonia and the mother disappeared, so it was impossible to follow up the history.

In another case, a child had been under treatment in the nervous clinic of the University Hospital for spastic diplegia due to hereditary lues. As there was no thought of an eye condition, the child was not referred to the eye clinic. The Wassermann was faintly positive for the baby and the father; the mother's was negative. The child was given mercury but did not improve. I saw the patient when two years five months old. The macular picture was lacking, and the vessels were almost normal in size, but the discs were white. The child was blind and idiotic. The spasticity of the lower limbs persisted, although the rest of the body was flaccid. A second child, then six months old, was healthy.

DR. WILLIAM EVANS BRUNER (Cleveland, Ohio): I desire to add merely a few words in regard to one point brought out by Mr. Collins—*x-ray* examination in hereditary optic atrophy. The essayist refers to the work of Mr. Fisher along this line. His results were first published in 1916. In 1912, or four years earlier, I read a paper before the American Ophthalmological

Society upon "Hereditary Optic Atrophy, with X-ray Findings."¹ The paper pretends to give no new theory, but does show some points of interest and especially one—the x-ray findings which suggest the advisability of future study along this line in other cases of the same and allied diseases.

The patient, a man aged thirty-six, was seen by Dr. de Schweinitz, who concurred in the diagnosis and wrote: "I do not recall that I have before x-rayed a patient with this type of optic nerve atrophy, but certainly the stereoscopic x-ray plates show marked thickening either in the sphenoid region or in the neighborhood of the sella, and it occurs to me that this lesion, whatever it may be, has something to do with the optic nerve disease."

Dr. Spiller, of Philadelphia, made a neurologic examination, confirmed the diagnosis and adds: "The x-rays show a projection upward of the floor of the sella turcica and suggest that the pituitary body may have its function impaired in this way. I do not know in how many cases of family optic atrophy x-ray plates have been made."

X-ray plates were made of a sister who had the disease and of a nephew who from the history also probably had it. By way of comparison, plates were made also of his mother and a sister who did not have the disease, and their plates showed nothing abnormal.

Later he saw Dr. Harvey Cushing, who also agreed with the diagnosis, though the subsequent history would indicate that it was an atypical case.

I closed the paper with the "hope that other patients presenting similar symptoms may be subjected to like x-ray examinations." I have not had the opportunity of seeing another case of this disease since that time and have naturally been much interested in the results of such examinations by other observers.

MR. E. TREACHER COLLINS (closing): First of all I would like to congratulate Prof. Finlay on having such a fine field for pathologic investigation of hereditary diseases as Cuba. I hope we may soon hear from him of a case of hereditary optic atrophy on which he has been able to perform a pathologic examination. I am also interested in his cases of degeneration of the cornea in which some members had the nodular and others the lattice-like type. That helps to confirm the theory that the two are but different manifestations of the same affection.

Dr. Verhoeff takes exception to the term "abiotrophy"—he prefers "premature senility." Premature senility seems to me to imply something to do with old age, while some of the cases I brought before you began quite early in life, for instance, the amaurotic family idiocy. It does not seem correct to speak of senility, even premature senility, in connection with a disease beginning early in life. Again, the term "abiotrophy" goes farther and gives more the idea that Sir William Gowers had in mind in using it—that is, a loss of some vital energy. Dr. Verhoeff also takes exception to the hyaline substance met with on the inner surface of the choroid being regarded as a degeneration. He regards it as due to proliferation. The condition is most certainly a degeneration in function even if the cells do not absolutely perish.

¹ Trans. American Ophthalmological Society, 1912; and Archives of Ophthalmology, 1912, vol. xl, p. 435.

Dr. Blaauw, I am glad to hear, agrees with me in liking the term "abiotrophy." He also agrees that the senile cataract begins in the cortex.

Dr. Libby says the recognition of these cases helps us in our statements to patients. I quite agree with that. Since I came to regard these cases from my present point of view it had given me much confidence in speaking to patients as to prognosis and of those precautionary measures they should take in regard to nutrition, etc. I feel I can speak with more confidence.

Dr. Jackson compared retinitis pigmentosa with a syphilitic choroidoretinitis. They are often very similar ophthalmoscopically. We know that in syphilitic choroiditis the primary change is vascular; it is a change in the capillary layers producing secondary degeneration of the rods and cones, not a primary degeneration of those structures. I congratulate Dr. Jackson on his optimistic outlook in treatment of these conditions.

I think Dr. Buchanan's case must have been one of amaurotic family idiocy from the account she gives of it.

Dr. Bruner spoke of *x*-ray findings in a case of hereditary optic atrophy. I quote a number of cases in my paper, and have learned from Dr. Zentmayer that he also reported a case, some of them with slight changes, and others without. In cases of abiotrophy we should expect overgrowth of fibrous tissue. The changes seen in the *x*-ray picture might be due to a thickening of the chiasma pressing down on the pituitary body instead of the pituitary body pressing up on the chiasma.

BITEMPORAL CONTRACTION OF VISUAL FIELDS IN PREGNANCY

PROF. C. E. FINLAY

Havana, Cuba

On February 15, 1919, a case was sent to me in consultation which gave rise to the investigations which form the subject of this paper.

Its clinical history was as follows:

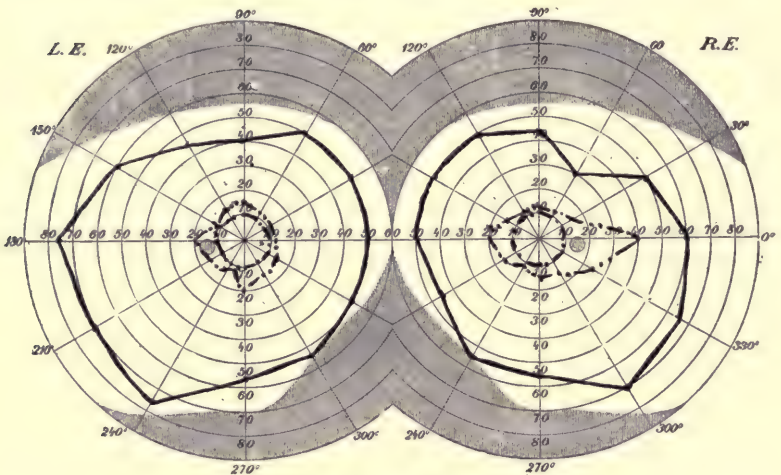


Fig. 1.—E. P. M., February 15, 1919.

Mrs. E. P. M., aged twenty-four years, a leading society lady, in the eighth month of pregnancy, had suffered a month previously a severe attack of influenza. After recovery she had developed a series of seizures of a cataleptic nature, during which she could not move, though perfectly conscious of what was going on around her. My examination of her eyes resulted as follows:

Corneæ and conjunctivæ normal.

Media clear.

Pupillary reactions normal.

Fundi: Slight contraction of retinal arteries and dilatation of retinal veins.

Visual acuity (each), 20/20.

Visual fields: Temporal contraction (more pronounced on right side) for white, with marked concentric contraction for colors, the red field being larger than that for blue, there being also a crossing of the limits of the color fields on the left side (Fig. 1).

I concluded that there exists a hypophyseal compression of the chiasm, but was at a loss as to whether this was due to a hypophyseal tumor or to an enlargement of the hypophysis in connection with pregnancy, and as to whether the cataleptic seizures were due to the enlargement of the hypophysis or of a hysteric nature. The further history of this case has no particular bearing on this paper. The seizures continued after confinement, and some of the experts who later saw the patient thought there was some endocrinic disturbance with hysteric manifestations. But the study of the case made me seek to determine whether the normal hypertrophy of the pituitary body which takes place in connection with pregnancy was of such a

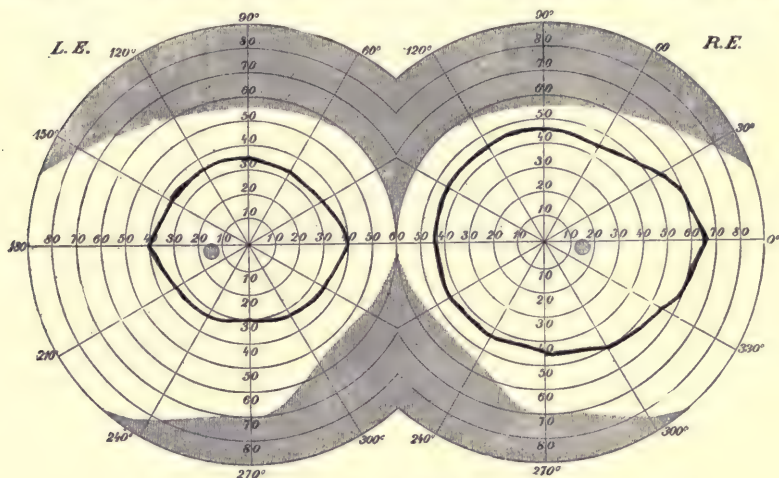


Fig. 2.—Case I. E. E., July 10, 1920.

degree as to determine changes in the visual fields. Through the courtesy of Professor Bustamante, in charge of the Maternity Clinic of the General Calixto Garcia Hospital, I proceeded to examine the visual fields of a number of women awaiting confinement, selecting only cases that presented no general complications, with normal eye-grounds and perfect visual acuity.

The following are the reports of the 31 cases examined:

CASE I.—E. E., white, aged twenty, Cuban. Pregnancy, eight months.

Urine normal.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Marked contraction—more marked in temporal half; more pronounced on left side (Fig. 2).

CASE III.—J. F., Cuban, mulatto, aged thirty-six. Nine months pregnant.

Urine: No albumin; specific gravity 1000.

Visual acuity (each), 20/20.

Visual fields: Marked bitemporal contraction (Fig. 3). This patient had a normal labor on August 16, 1920, and a second examination ten days later showed a slight reduction in the bitemporal defects.

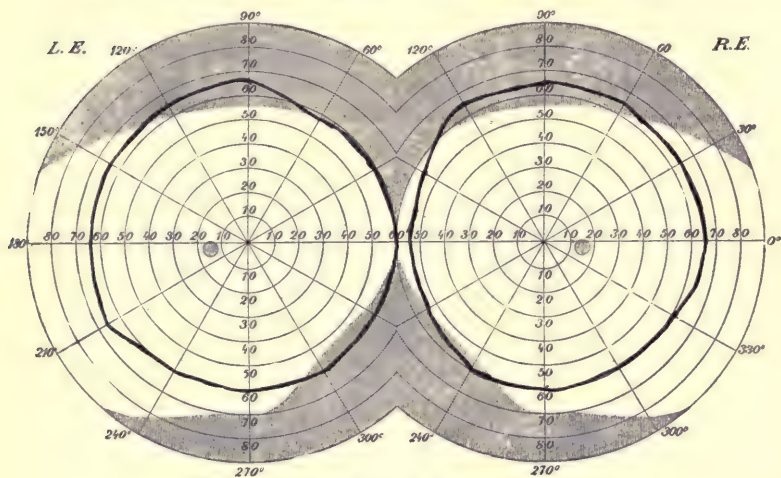


Fig. 3.—Case III. J. F., July 10, 1920.

CASE IV.—L. A., Cuban, negress, aged sixteen. Nine months pregnant.

Urine: No albumin; specific gravity 1011.

Fundi normal. Visual acuity (each), 20/20.

Visual fields: Marked bitemporal contraction (Fig. 4).

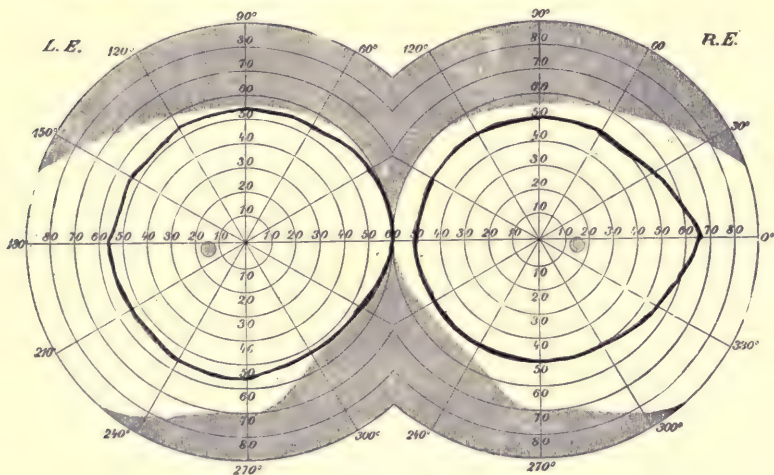


Fig. 4.—Case IV. L. A., July 13, 1920.

CASE VII.—C. C., Cuban, mulatto, aged twenty-nine. Eight months pregnant.

Urine: Trace albumin; specific gravity 1020.

Fundi normal. Visual acuity (each), 20/20.

Visual fields: Very pronounced concentric contraction, greater in temporal half (Fig. 5).

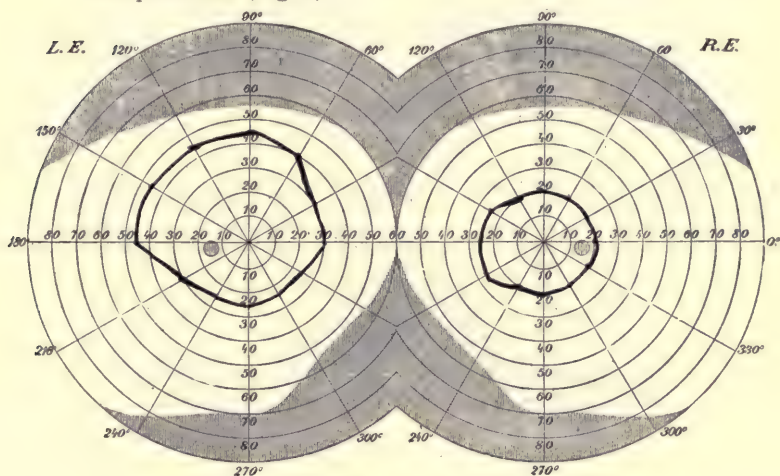


Fig. 5.—Case VII. C. C., July 19, 1920.

CASE VIII.—C. V., Cuban, mulatto, aged thirty-seven. Nine months pregnant.

Urine: No albumin; specific gravity 1031.

Fundi normal. Visual acuity (each), 20/20.

Visual fields: Marked bitemporal contraction (Fig. 6).

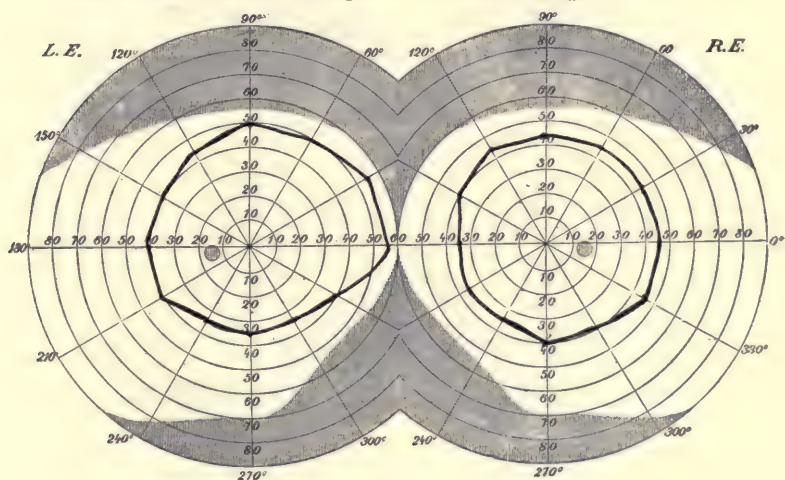


Fig. 6.—Case VIII. C. V., July 15, 1921.

CASE X.—M. C., white, aged thirty-eight. Eight months pregnant.

Urine: No albumin; specific gravity 1024.

Fundi normal. Visual acuity (each), 20/20.

Visual fields: Marked bitemporal contraction (Fig. 7).

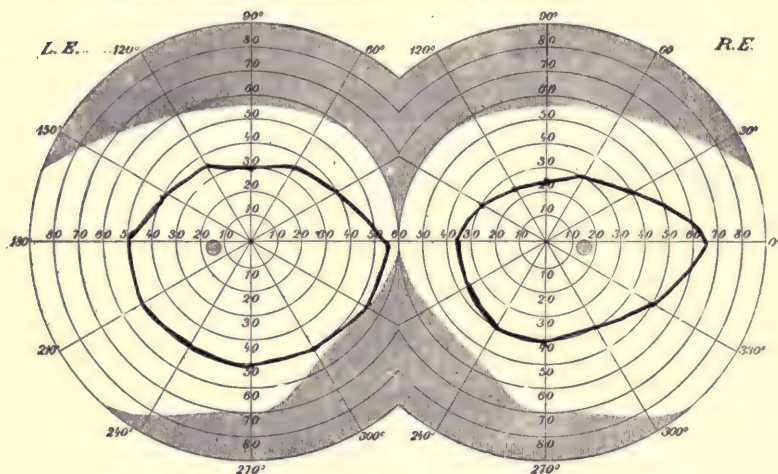


Fig. 7.—Case X. M. C., July 17, 1921.

CASE XI.—C. G., Cuban, mulatto, aged twenty-eight. Eight months pregnant.

Urine: No albumin; specific gravity 1016.

Fundi normal. Visual acuity, 20/20.

Visual fields: Moderate bitemporal contraction (Fig. 8).

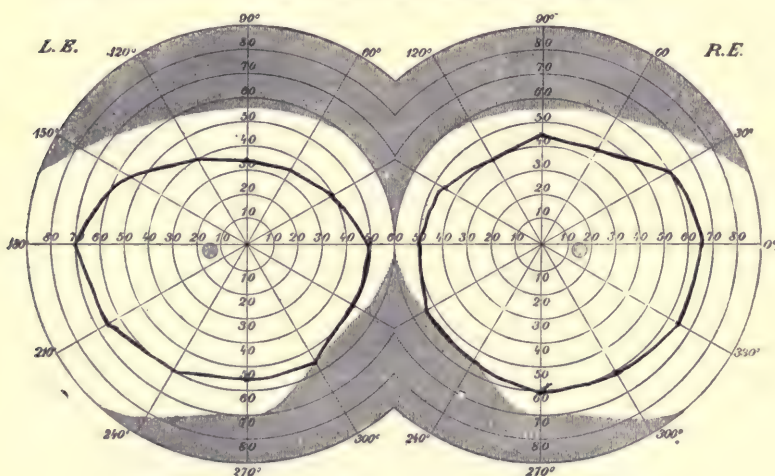


Fig. 8.—Case XI. C. G., July 17, 1921.

CASE XIII.—M. C., Cuban, white, aged eighteen. Nine months pregnant.
 Urine: Trace albumin; specific gravity 1012.
 Fundi normal. Visual acuity (each), 20/30.
 Visual fields: Marked bitemporal contraction (Fig. 9).

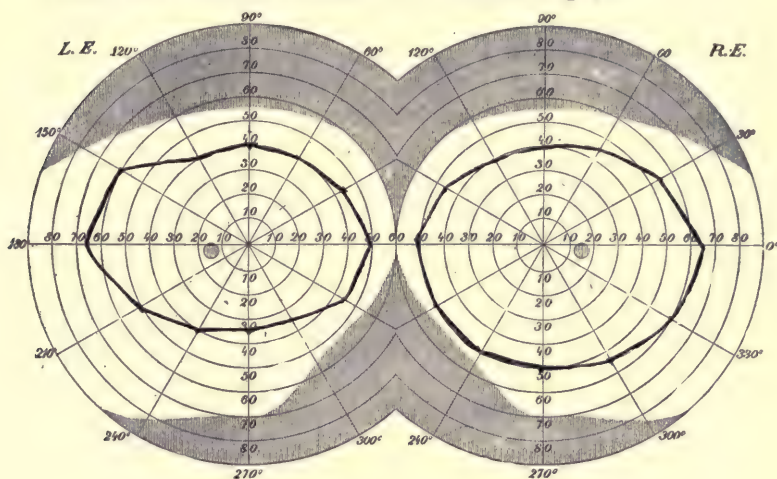


Fig. 9.—Case XIII. M. C., July 22, 1921.

CASE XIV.—A. I., Cuban, negress, aged seventeen. Eight months pregnant.
 Urine: No albumin; specific gravity 1012.
 Fundi normal. Visual acuity (each), 20/20.
 Visual fields: Moderate bitemporal contraction (Fig. 10).

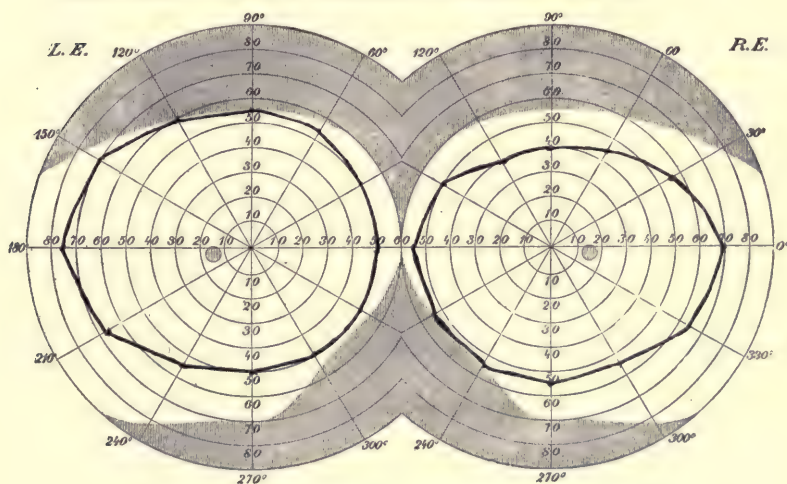


Fig. 10.—Case XIV. A. I., August 21, 1921.

CASE II.—J. G., white, Spanish, aged twenty-two. Nine months pregnant.

Urine: No albumin; specific gravity 1020.

Fundi normal.

Visual acuity (each), 20/20.

Visual fields: Practically normal.

CASE V.—A. G., Cuban, negress, aged sixteen. Seven months pregnant.

Urine: Trace albumin; specific gravity 1015.

Fundi normal.

Visual acuity (each), 20/20.

Visual fields: Bitemporal contraction.

CASE VI.—R. G., Cuban, white, aged twenty-one. Nine months pregnant.

Urine: Trace albumin; specific gravity 1023.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Very slight contraction (R); slight contraction (L).

CASE IX.—G. S., Cuban, negress, aged thirty-seven. Nine months pregnant.

Urine: No albumin; specific gravity 1018.

Fundi normal.

Visual acuity (each), 20/20.

Visual fields: Moderate bitemporal contraction.

CASE XII.—M. S., Cuban, white, aged fourteen. Five months pregnant.

Urine: Trace albumin; specific gravity 1016.

Fundi normal.

Visual acuity (each), 20/20.

Visual fields: Slight temporal contraction (R); moderate temporal contraction (L).

CASE XV.—F. A., white, aged twenty-three. Nine months pregnant.

Urine: Trace albumin; specific gravity 1030.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Practically normal.

CASE XVI.—S. A., white, aged twenty-five. Nine months pregnant.

Urine: No albumin; specific gravity 1022.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XVII.—V. A., Cuban, negress, aged eighteen. Nine months pregnant.

Urine: No albumin; specific gravity 1005.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XVIII.—Cuban, mulatto, aged thirty-one. Nine months pregnant.

Urine: No albumin; specific gravity 1030.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Practically normal.

CASE XIX.—C. H., Cuban, negress, aged twenty-eight. Nine months pregnant.

Urine: No albumin; specific gravity 1012.

Fundi normal.

Visual acuity, 20/20.

Visual fields normal.

CASE XX.—G. G., Cuban, negress, aged thirty-one. Nine months pregnant.

Urine normal.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XXI.—J. B., Cuban, mulatto, aged thirty-nine. Nine months pregnant.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Moderate bitemporal contraction.

CASE XXII.—F. I., Spanish, aged twenty-five. Nine months pregnant.

Urine: No albumin; specific gravity 1012.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XXIII.—C. F., Cuban, negress, aged sixteen. Nine months pregnant.

Urine: No albumin; specific gravity 1010.

Visual acuity, 20/20.

Visual fields: Slight temporal contraction (R).

CASE XXIV.—P. R., Spanish, aged thirty-six. Eight months pregnant.

Urine: Trace albumin; specific gravity 1023.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight temporal contraction (L).

CASE XXV.—B. P., Cuban, mulatto, aged eighteen. Eight months pregnant.

Urine: No albumin; specific gravity 1012.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XXVI.—M. O., Spanish, white, aged twenty-two. Eight months pregnant.

Urine: Trace of albumin; specific gravity 1020.

Fundi normal.

Visual acuity, 20/20.

Visual fields: Slight bitemporal contraction.

CASE XXVII.—A. A., Spanish, white, aged thirty-four. Nine months pregnant.

Urine: No albumin; specific gravity 1026.

Fundi normal.

Visual acuity, 20/20.

Visual fields normal.

CASE XXVIII.—V. S., Cuban, negress, aged twenty-two. Nine months pregnant.

Urine: Trace albumin; specific gravity 1018.

Fundi normal.

Visual acuity, 20/20.

Visual fields normal.

CASE XXIX.—C. L. V., Cuban, white, aged seventeen. Eight months pregnant.

Urine: No albumin; specific gravity 1025.

Fundi normal.

Visual acuity, 20/20.

Visual fields practically normal.

CASE XXX.—A. P., Cuban mulatto, aged nineteen. Nine months pregnant.

Urine: No albumin; specific gravity 1016.

Fundi normal.

Visual acuity, 20/20.

Visual fields normal.

CASE XXXI.—F. P., white, aged twenty-eight. Eight months pregnant.

Urine: Trace albumin; specific gravity 1031.

Fundi normal.

Visual acuity, 20/20.

Visual fields normal.

From an examination of these visual fields we find that out of the 31 cases examined only 9 could be considered approximately normal, the remainder showing changes in the nature of a temporal contraction; of these, 8 were slight, 9 moderate, and 5 pronounced. These surprising results led me to a thorough study of the literature at hand in connection with the anatomic and other data requisite for a complete explanation. For a long time I was under the impression

that my observations were the only ones made in this connection, but I eventually discovered that changes in the visual fields of a similar nature to those which I have recorded have been published first by Bellinzona and Tridonani,¹ and later confirmed by Forti,² which they all attributed to dynamic or vasomotor disturbances similar to those occurring in hysteria.

This more or less forced explanation seems to me unnecessary, as the changes described can be perfectly explained by the mechanical pressure brought to bear on the chiasm by the enlarged pituitary gland resulting from the normal hypertrophy of this organ which occurs in pregnancy. The gland increases two to three times its weight and volume during this state,³ this being due to an increase in number of oxyphil cells (*Schwangerschaftszellen*) in the "pars anterior,"⁴ a good many of the disturbances occurring in connection with pregnancy being probably due to the corresponding hyperfunction. The chiasm, which is situated over the center of the "sella turcica," and not in contact with its anterior border, from which it may be pressed upon by a reflexion of the dura mater (*cisterna chiasmatis*) from behind and below by an enlarged hypophysis, if this attains at least a volume of 0.5 cm.

The differences in the degree of the changes in the visual fields depend on the degree of hypertrophy and on anatomic peculiarities which may favor or hinder the above-mentioned compression.

In conclusion I consider I can establish: That during pregnancy there often occurs, as a result of the normal hypertrophy of the pituitary gland in connection with this state, a compression of the chiasm which manifests itself by changes in the visual fields in the nature of a bitemporal contraction which varies in degree according to the amount of compression suffered, this depending on the corresponding amount of hypertrophy and on the anatomic peculiarities of the case which may favor or hinder this compression.

DISCUSSION

DR. W. B. LANCASTER (Boston, Mass.): Professor Finlay's proposition has a great deal of theoretical interest. In order to establish his thesis two points have to be proved: first, that bitemporal contraction of the visual field is fairly constant in the normal pregnant woman; second, that enlargement of

¹ Bellinzona and Tridonani: *Boll. d. Soc. med.-chir. di Pavia*, 1903.

² Forti: *Arch. di ottal.*, February, 1910.

³ Shafer: *Endocrine Glands*, London, 1916, p. 115.

⁴ Erdheim and Stumme and Emory Hill: *Amer. Encyc. Ophth.*, xiii, 10232.

the pituitary is also a fairly constant phenomenon in the normal pregnant woman. As to the latter point, various men long ago surmised that the pituitary was enlarged in pregnancy, notably Le Conte in 1898, and Launois and Mulon in 1903. In 1908 Erdheim and Stumme demonstrated the incidence of this enlargement by the examination of 150 subjects, and they showed that in women who had never been pregnant the average weight of the hypophysis was 61.8, the maximum, 75; in primiparæ the average was 84.7, the maximum, 110; in multiparæ the average was 106, the maximum, 165. This shows beyond reasonable doubt that there is a normal enlargement of the pituitary in the pregnant woman.

As to the second point, I happened to be so situated as to have material well suited to add to the data. It is just a question of collecting a sufficient number of fields from a sufficient number of independent sources to establish or disprove the thesis. I was unable personally to make these tests, but I was fortunate in enlisting the services of Dr. Maud Carvill, who is my associate on the staff of the New England Hospital for Women and Children. They have a large obstetrical clinic well suited to this sort of test. One always wishes that he could himself examine the fields when a doubtful point comes up, but I think if you will examine these charts,—and several hundred have been taken,—you will see that they bear in themselves the marks of genuine, accurate, competent work.

If you, therefore, can establish the thesis that the pituitary is enlarged in normal cases, and that the visual fields are bitemporally contracted, the conclusion is irresistible that one is the cause of the other, just as various other phenomena of pregnancy can be attributed to this enlargement of the hypophysis, which is not always free from pathologic consequences.

DR. MAUD CARVILL (Boston, Mass.): Through the courtesy of the staff of the Maternity Department of the New England Hospital for Women and Children of Boston we are making a study of a group of women in the later weeks of normal pregnancies to learn to what extent the hypertrophy of the hypophysis occurring in pregnancy affects the visual fields.

Up to the present time we have examined the visual fields of sixty-seven gravid women, the progress of whose pregnancies was otherwise normal, *i. e.*, there were no complications. Their blood pressures, urines and general conditions were considered normal by the internist. Their fundi were normal, and their vision was of normal acuity. These women were of good intelligence, many of them former students in our high schools and some graduates therefrom. Their central fixation was steady and their peripheral tests very accurate. The definiteness of the delineation of the fields has impressed us. In many cases the variation of a degree in the position of the test object was a definite question of seeing it or not seeing it. The fields were taken by daylight by a northerly exposure, with a perimeter with a radius of 28 cm., using a 5 mm. opaque white test object.

Six of these sixty-seven cases we are not including, as their last examination was taken more than one month before parturition and at that time their fields were normal. Of the remaining sixty-one cases, only seven, or 11 per cent., could be considered normal; the remaining fifty-four cases, or 89 per

cent., showed bitemporal contraction of greater or less extent; sixteen cases, or 28 per cent., showed a marked contraction (20° or more); twelve cases, or 20 per cent., showed moderate contraction (10° to 20°); twenty-six cases, or 42 per cent., showed slight contraction (less than 10°). Five degrees was the minimum regarded as a definite contraction.

The cases we have noted in the literature refer to the multiparous gravid state. Professor Finlay has not stated the number of pregnancies in his cases. About 57 per cent. of our cases were primiparæ.

TABLE SHOWING THE VARYING DEGREE OF CONTRACTION ARRANGED BY GRAVID STATE

Contraction	Pregnancies								
	I	II	III	IV	V	VI	VII	VIII	IX
16-28% marked	11	1	..	2	..	1	1
12-20% moderate	4	6	1	1
26-42% slight	15	2	4	2	..	1	2
7-11% normal	5	1	1
Total	35	10	4	4	..	2	3	1	2

TABLE SHOWING VARYING DEGREES OF CONTRACTION OF FIELDS ARRANGED BY AGE OF PATIENT

Contraction	Age														
	18	19	20	21	22	23	24	25	26	27	28	29	30	35-40	40-44
Marked	1	..	1	..	3	3	1	2	1	1	..	2
Moderate	1	1	..	5	2	2	5	3	6	1	..
Slight	1	1	1	..	1	1	1	1	2	..	1
Normal	1	..	1	..	1	..	1	..	1	1	1	1	..	1	..

TABLE SHOWING FIELDS IN WEEKS PRECEDING PARTURITION

Period	Contracted	Marked	Moderate	Slight	Normal
One week before	53	14	17	22	13
Two weeks before	35	8	10	17	8
Three weeks before	24	7	8	9	7
Four weeks before	15	4	6	5	6
Five weeks before	10	2	5	3	5
Six or more weeks before	4	1	2	1	3

The conclusive studies of Erdheim and Stumme prove the question of functional hypertrophy beyond the stage of conjecture. These authors have demonstrated by actual measurement an increase in size and weight of the structure of the pituitary gland. After parturition there occurs a subsidence, the involution being complete at the termination of lactation. With a succeeding pregnancy a further augmentation takes place. Bandler states in his book on "Endocrinology," published in 1921, that "the gland never goes back to its former ante-pregnant stage."

In the 42 cases which we have examined postpartum, all but four have had normal fields. We think later examinations would have found these normal.

PROFESSOR F. DE LAPERSONNE (Paris, France): J'ai été extrêmement intéressé par la communication de M. le Professeur Finlay. Si ses recherches sont confirmées dans un très grand nombre de cas, c'est un nouveau chapitre qui s'ouvre pour la physiologie pathologique de l'hypophyse. Dans les champs visuels qui nous sont présentés, tous n'ont pas les dispositions caractéristiques de l'hémianopsie bitemporale.

A côté de ces troubles passagers, disparaissant après la grossesse, il en est peut-être qui persistent. J'ai observé une dame de 35 ans qui, au cours de sa troisième grossesse, eut des céphalées, de la polyurie, sans glycosurie, et différents troubles endocriniens. L'ayant examinée huit mois après la grossesse, je constatai une hémianopsie bitemporale avec atrophie partielle de la papille; l'autre oeil était atrophie depuis longtemps. Le Wassermann était négatif et la selle turcique légèrement augmentée. Si la grossesse n'a pas été la cause primaire de cette lésion hypophysaire, elle paraît l'avoir nettement aggravée.

PROFESSOR C. E. FINLAY (closing): In regard to Dr. Lancaster, I am glad he is able to confirm both the enlargement of the gland, as well as the bitemporal contraction. I am interested in seeing the cases of Dr. Carvill and how the visual fields compare with mine, especially as Dr. Lancaster mentions that some of these cases are quite typical in their characteristics. Owing to the length of time of pregnancy the bony body where the hypophysis lies is hardly formed, and one would not expect it except where there is some compression against a bony wall.

I have had a similar case to that mentioned by Professor de Lapersonne of the appearance of these symptoms after pregnancy. Most of the cases which I followed afterward showed that the bitemporal contraction disappeared comparatively rapidly after pregnancy, but lately I saw a woman who, two years after pregnancy, was suffering from headaches and blurring of vision, and in whom I found a slight optic neuritis and a bitemporal contraction. I cannot state whether this was the hypertrophy of pregnancy which had not disappeared, or whether the pregnancy provoked this in a patient already predisposed, or whether it was simply an independent hypophyseal tumor. But examination showed a marked enlargement of the sella turcica.

LES ÉCHANGES D'EAU ET D'IONS À TRAVERS LA CORNÉE

DR. V. MORAX

Paris, France

La question des échanges à travers la cornée est une de celle qui doit intéresser le plus l'ophtalmologiste puisque la part la plus importante de sa thérapeutique locale repose sur les phénomènes d'absorption et d'absorption qui se produisent à travers la coque oculaire. Lorsque par une instillation d'atrophine ou de pilocarpine, nous provoquons la dilatation ou le rétrécissement de la pupille, nous faisons passer dans la chambre antérieure une certaine quantité de substance active, celle-ci étant à son tour absorbée par le tissu irien.

Si cet effet physiologique du passage des substances médicamenteuses est bien connu, le mécanisme de sa production est par contre très ignoré. Ce n'est certes pas que la question n'ait pas été étudiée de divers côtés mais elle l'a été surtout jusqu'ici au point de vue purement chimique. Or, l'analyse chimico-physique a transformé l'étude des échanges à travers les membranes.

De nouvelles conceptions sur la tension osmotiques des solutions, sur la constitution ionique, des sels se sont développées et il nous a paru nécessaire d'aborder la question des échanges à travers la cornée à la faveur de tous les progrès réalisés dans l'étude des phénomènes chimico-physiques. Les recherches dont je voudrais donner ici un court résumé ont été faites en collaboration avec M. Girard ou avec M. Girard et Mestrézat. Bien qu'elles soient encore loin d'être achevées, certains résultats peuvent dès maintenant intéresser l'oculiste et le physiologiste.

Il s'attache en effet un grand intérêt pratique à l'étude des moyens qui permettent de modifier le "milieu intérieur de l'œil qui est l'humeur aqueuse. Ces modifications peuvent être envisagées à deux points de vue; au point de vue du volume liquide dont dépend un facteur physiologique essentiel qui est la tension oculaire et au point de la constitution chimique et notamment de la constitution ionique de l'humeur aqueuse. Ce dernier problème qui touche également à

la thérapeutique, est en somme celui de la perméabilité de la cornée aux substances dissoutes dans les solutions qui en baignent l'épithélium externe.

Nos recherches ont d'abord trait à un moyen pratique nouveau de faire varier le volume de l'humeur aqueuse dans la chambre antérieure et de modifier par conséquent la tension oculaire en élevant ou en abaissant considérablement la valeur. Ce moyen, c'est l'osmose électrique (endosmose ou exosmose). Nous ne voulons pas développer ici la théorie du phénomène et la représentation qu'on peut se faire de son mécanisme; nous ne l'encisagerons que comme moyen pratique. Disons d'abord que ces osmose électriques, nous les avons pratiqués sur l'œil vivant et en place de l'animal vivant. Essentiellement, elles consistent dans le glissement sous l'action d'un champ électrique, de veines liquides dans les interstices cellulaires d'un tissu vivant,—la cornée en l'espèce. L'un des pôles de la source électrique qu'on utilise, étant relié à un point quelconque du corps de l'animal par une large électrode, une électrode en platine reliée à l'autre pôle plonge dans la solution d'électrolyte dont est baigné par le moyen d'un bain d'œil en verre l'épithélium externe de la cornée. La théorie du phénomène fait prévoir que pour une valeur et une orientation fixées du champ—différence de potentiel entre la cornée et la solution qui en baigne l'épithélium externe—ce sera de la constitution ionique de cette solution que dépendra le sens du glissement des veines liquides dans les interstices cellulaires: endosmose ou exosmose.

Pour une connection polaire telle que la cathode étant au corps de l'animal l'anode plonge dans le liquide du bain d'œil, on obtient lorsque celui-ci est constitué par des solutions de sulfate de magnésie des endosmose qui font monter la tension intraoculaire, au bout de 30 minutes environ de 25 à 70 ou 50 millimètres de Hg. Au contraire, toujours pour la même connection polaire, lorsque ce liquide est constitué par une solution de nitrate de calcium, cette tension oculaire s'abaisse dans le même temps du fait de l'exosmose de l'humeur aqueuse qui filtre à travers la cornée hors de l'organisme à 8 ou 10 millimètres de Hg.

A la fin de l'expérience, lorsque les précautions nécessaires ont été prises (concentrations moléculaires convenable de la solution, débit électrique ne dépassant pas 2 milliampères et surtout absence d'électrolyse), on ne constate aucune lésion de la cornée, non plus qu'aucune trace d'œdème.

Ajoutons que sur les yeux dont la circulation est normale et sur lesquels aucun blocage vasculaire n'a été pratiqué, ces déséquilibres de tension se réparent au bout d'une heure environ et l'humeur aqueuse dite de nouvelle formation diffère alors de l'humeur aqueuse normale.

Mais ce n'est pas seulement le volume de l'HA., c'est aussi la constitution ionique qu'il est possible de modifier momentanément tout au moins.

L'utilisation d'un champ électrique pourra être, en vue de cette fin très avantageuse; mais elle n'est pas indispensable et au cours de nos expériences sur les échanges d'ions à travers la cornée, nous avons utilisé très souvent la diffusion pure et simple sans source électrique extérieure.

Le fait essentiel que nous avons trouvé—en collaboration pour cette partie avec M. Mestrézat—peut se résumer ainsi: lorsqu'une solution d'un sel détermine, dissocié en ses ions, baigne la cornée dans l'HA., ce n'est pas le sel qui diffuse à travers la cornée dans l'humeur aqueuse, ou plus exactement les deux ions de ce sel, ainsi dissocié ne diffusent jamais en proportions chimiquement équivalentes. Si, par exemple, la solution est constituée par du sulfate de magnésie, il ne diffusera pas dans l'humeur aqueuse, un nombre égal d'anions et de cations (comme cela se passerait si c'était du sulfate de magnésie qui diffusait dans la chambre antérieure, mais il diffusera beaucoup plus d'ions magnésium que d'ions sulfate en sorte que ce ne sera plus du sulfate de magnésie qui aura diffusé dans l'humeur aqueuse. Mais d'autre part, comme l'excès de magnésium a diffusé sous forme d'ions porteurs de charges électriques positives, et que l'humeur aqueuse reste cependant électriquement neutre—aucune charge non compensée n'y apparaissant le principe de l'équilibre électrique exige qu'au passage dans un sens d'un excès d'ions, corresponde le passage en sens inverse—c'est à dire vers la solution baignant l'œil—d'un nombre électriquement et chimiquement équivalent d'ions (métalliques) de même signe. Ce serait, par exemple, les ions sodium de l'humeur aqueuse.

Ensorte que le contact de l'épithélium externe de la cornée avec une solution de sulfate de magnésie—nous prenons ce sel à titre d'exemple—n'a pas du tout, comme conséquence comme il eut été naturel de le penser, l'enrichissement de l'humeur aqueuse en sulfate de magnésie; l'humeur aqueuse s'enrichit relativement beaucoup en magnésium, très peu en ions SO_4 —Pour certains sels, comme les sels d'ammonium, la discrimination est tellement accusée que pratique-

ment un seul ion passe—et elle s'appauvrit en un métal qui sera par exemple du sodium en une proportion électriquement et chimiquement équivalente à celle en laquelle elle s'est enrichie en magnésie. Il en résulte dans la composition ionique de l'humeur aqueuse de très importantes modifications.

Pour certains sels comme les sels monovalents d'ammonium ce sont au contraire les anions (radicaux acides) qui diffusent en excès, excès si grand parfois qu'on peut ici parler d'une véritable hemiperméabilité ionique. D'ailleurs il est possible en faisant varier la réaction du milieu (légère alcalinité ou légère acidité) de modifier ou même d'inverser le sens de ces résultats.

La réelle difficulté dans ces expériences a été de pratiquer sur des quantités très petites (chaque ponction d'humeur aqueuse ne donnant guère que 2 à 3/10 de cent.) le dosage des 2 ions de chaque sels. Seules les méthodes microchimiques permirent de la surmonter. De l'enquête systématique que nous comptons poursuivre relativement à cette perméabilité élective de la cornée vis à vis lesions, sortiront peut-être d'intéressantes conclusions chimio-thérapeutique.

PSYCHOPATHOLOGY IN OPHTHALMIC PRACTICE

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Ophthalmology occupies, in many ways, a central position among the various departments of medical and surgical practice. This was well shown in a paper by Dr. F. Park Lewis at the meeting of the American Medical Association at Atlantic City, N. J., in June, 1919. The author took as an illustration the close connection between dental caries and many forms of ophthalmic disease. He might have greatly extended the scope of his paper and traced the intimate connection of ophthalmology with all the general infections, especially those which are of a chronic character.

There is, however, a new field of medical research which is rightly claiming close attention at the present time which is destined to influence every branch of medical study, and that is the rapidly developing science of psychopathology. No department is more likely to be affected by this than that of ophthalmology. It is not only that ophthalmic conditions often form an exciting factor in the onset of the psychoneuroses, but that there are psychic elements com-

plicating most ophthalmic diseases. Thus again we find the central place of ophthalmology and the necessity of recognizing the psychologic aspects of ophthalmic work, and, where necessary, of full coöperation with those who practise psychotherapy.

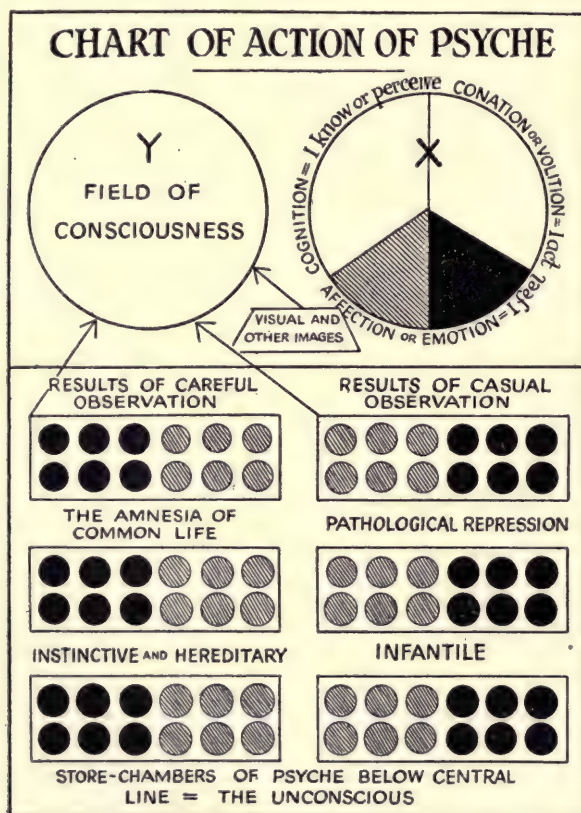
I have been led to realize the urgency of this problem by certain circumstances which it may be of some interest to record. During the war, when acting as an ophthalmologist in connection with a British division composed very largely of miners, I was for the first time brought in contact with the ailment most unfortunately named miners' nystagmus. Recognizing the seriousness of the malady, and believing that it would be of some value to record observations on these cases when removed from underground work, I prepared a paper on this subject. The paper was published in the *British Medical Journal* on March 4, 1916, with the title, "Visual Neuroses of Miners in Their Relation to Military Service." I pointed out that nystagmus was only one of a series of symptoms in the disease, and drew attention to the great importance of the mental factor and the possibility that these cases might be treated by suggestion. At that time I was not interested in psychology as I have been during the past two years, but the study of these cases gave me a special interest in the relation between the mind and vision.

At the Ophthalmological Congress in London in May, 1921, I took part in a discussion on the Psychology of Vision in Health and Disease, and expressed in that my strong conviction as to the importance of knowledge of modern psychologic teaching in ophthalmic work. I had for the previous eighteen months been engaged in special study on these lines, with the result that I had benefited in my own powers of work and found great scope for the exercise of the knowledge gained in my own practice.

After six months' further study I decided to formulate more clearly the conceptions which had helped me, and on November 11, 1921, I contributed a paper to the Ophthalmological Section of the Royal Society of Medicine of London on "The New Psychology in its Relation to Problems of Vision." This has been published in the *Medical Press and Circular* on November 30 and December 7, 1921. The present paper is intended to be a sequel, and it will be necessary to refer very briefly to the line adopted in that contribution, seeing that a proper appreciation of the general lines of normal psychology is essential before proceeding to deal with psychopathology. This is the more necessary in view of the great diversity of opinion which prevails on

these subjects. A chart was used to represent the working of the psyche, and this is reproduced here in order to make clear the standpoint of the author. The chart includes the following parts:

1. A circle X representing, as if it were a gramophone, the mechanism of the psyche divided into its three sections of a *cognition*, by which an image or impression, visual or otherwise, is recognized; an



affection, which stands for the emotional result of cognition, which in its turn leads to a *conation* or action.

2. A second disc, Y, as if it were a gramophone record, stood for the field of consciousness, which would present to the psyche the visual and other images or impressions received from external objects or from the memory or from both combined.

3. The store-chambers of the psyche or of the memory were described as divided into six compartments, referred to as—(a) results of careful observation; (b) results of casual observation; (c) amnesia of common life; (d) pathologic repression; (e) infantile impressions; (f) instinctive and hereditary factors. These signified the author's idea of what is usually termed the unconscious, and the circles within them indicated the mental concepts stored in the memory which were spoken of as psychograms.

The salient features in this scheme may be explained as follows:

1. The part of the circle X marked with shading or black was taken to represent the emotional part of the psyche, which was also regarded as the center of force or energy in the working of the mind. This, in the author's view, was comparable to the "elan vital" of Bergson, the "libido" of Freud or Jung, or "the urge" of English-speaking psychologists, whatever their special conception might be as to the nature of this all-pervading force.

The marking in a similar manner of the psychograms stored up in the unconscious indicated that each mental concept is activated by this emotional force, with its conflicting character. This might be illustrated by ideas of attraction or repulsion, of light or darkness, of harmony or discord, of rest or pain, of hope or fear, or any other antagonistic forces. This is laid stress upon seeing that the conflict graphically described by Freud, which is an essential element in psycho-analysis, may be regarded as a prominent symptom in all kinds of disease. Some aspect of distress is a constant factor in every ailment, whether it is a malignant tumor, toothache, or an error of refraction, and it is this which we, whose duty it is to combat human ills, are called upon to alleviate.

2. The second point of importance to which attention is directed is to the medium, as shown by the disc Y, whereby diverse impressions or images, whether of the past or present, are harmonized and presented to the main mechanism of the psyche.

It was this harmonizing or reconciliation between new images and past memories, or in psychologic terms of apperception, which was the main point of the paper which demonstrated, by various instances, that visual memory is an essential feature in every visual concept. The instances given were the following: infantile vision; individuals blind from birth; the recognition of strange objects; the use of optical apparatus; the artistic eye; the athletic eye.

The meaning of the disc Y was also regarded as an illustration of

even wider significance. Though it is marked in the chart with the words "field of consciousness," yet it is suggested that there may be modifications of this idea which might represent the dream state or the hypnotic state, and might be labeled Y1 or Y2. Thus, on falling to sleep the change from Y1 to Y2 would occur, this dream state being one in which access appears to be attained to chambers of the psyche which are inaccessible to the normal conscious state. These different states are instances of the process of dissociation which is responsible for most of the abnormalities of mental life, and is represented by cases of so-called multiple personality, forms of drug intoxication, or actual insanity.

This may also explain the visual irregularity spoken of as "*déjà vu*," or false recognition, which has been the subject of many hypotheses. Bergson has devoted much attention to it, and it has been dealt with recently by Kinnier Wilson at the last Ophthalmological Congress in London in 1921, when he gave instances of this phenomenon in relation to epilepsy. Of these epileptic cases I have no personal knowledge, but I have recently conversed with a number of people who claim to have had this experience in ordinary life. In these cases I am strongly of opinion that the condition is due to a momentary day dream. Let me give an illustration of this.

A soldier in the recent war came to the top of Cassel Hill in France, near to the Flanders frontier. When he saw the view of the plain stretched before him he at once felt that he had been there before, and had seen precisely the same picture, though he knew that this was impossible. A similar story is told by others, most of whom would reject with scorn the idea that they were remembering a similar situation or had only seen a picture of the view. They are certain that the picture is identical. This I believe is due to a day-dream. For the moment when they are brought face to face with the new condition, some alternative Y disc must be in operation which receives the impression and is immediately replaced by the normal conscious disc, and the whole impression has thereupon been relegated to the store-chambers of the memory without appreciation of space or time. This is the explanation which satisfies me and has satisfied those with whom I have spoken who have themselves experienced the phenomenon which is infinitely more important.

3. The third essential feature of the chart, the store-chambers of the memory, represents not only the accumulation of ideas, but the storehouse of energy. Starting from instinctive and hereditary be-

ginnings with the psychic momentum, which has been acquired throughout life, it forms the wonderful conception of the mind which is being unfolded to us in what may be called the story of the unconscious. Here is the inexhaustible mine from which we may obtain the solution of many of life's riddles; this is the great dynamic force which is elusive to the impetuous promptings of the will, but can be influenced by suggestion or auto-suggestion; yet all of these are under the control directly or indirectly of the personality of the normal man, and for them he is responsible.

Thus we have endeavored to picture the working of the human mind, not as an isolated problem, but in its relation to vision. No attempt has been made to correlate in any exact manner the functions of the psyche and the soma, or yet the higher realms of the pneuma, but it is the author's firm belief that each of the domains of spirit soul and body must be explored in their relation to one another. Only thus can we satisfactorily treat the whole man—the patient who seeks our aid.

For the moment it is the psyche and soma which are under special consideration because of the new light which is being thrown upon the working of the psyche. So far as these are concerned we must regard the psyche as the directing force, and as the nearest approach to the essential ego that we have yet reached. This then is the new standpoint from which we must view our ophthalmic practice. We must be no less scrupulous in our methods of examining organic defects, or in the identification of cerebral or nervous lesions, but above all we must include in our reckoning the psychologic factor.

The application of the new psychology to ophthalmic practice will only gradually be realized, but it is hoped that the suggestions which have been made as to the methods of mind-working may help many to interpret some of the difficulties occurring in their own practice.

We will now endeavor to describe some of the cases in which psychopathology and ophthalmology meet. Most of these will include some measure of dissociation in which repression with amnesia will have a part, and in which often, in proportion to the amnesia, there will be some emotional distress. In all of these it is probable that an unconscious motive can be detected, and the demonstration of this may lead to the resolving of the conflict, the restoration of harmony, and relief from the distress.

We shall proceed to consider these cases in three main classes:

I. THE PSYCHOPATHOLOGY OF EVERY-DAY LIFE

This admirable phrase, used by Freud as the title of a volume, will form a good introduction to our subject. It is a mistake to regard all psychic disturbances as conditions which can be dealt with only by specialists in psychotherapy. The truth is that there is a psychic element in every malady, and that whether we know it or not we must deal with it if we are to help our patients. Every practical physician or surgeon recognizes this, though perhaps unconsciously, and an older generation with its stress on temperament, anticipated much that is being taught in a new way to-day. The manner of the older practitioner was studied more closely perhaps than at the present time, and the confident assurance of success in treatment can be regarded only as a method of suggestion. To-day we approach these problems with new light. In our ophthalmic clinics public or private patients come to us not so much because they know of any distinct disability, but because they are anxious about themselves.

Mysterious pains in the eye or head, floating bodies or clouds before the eyes, unusual appearances about the eyes or lids, are often regarded as threatening disaster, perhaps blindness. In many cases the anticipation of evil is more serious than any actual disease, and may even produce illness, and in some this foreboding is the only trouble with which we may have to deal.

Obviously, our duty is to eliminate any organic disease or cause for trouble, including particularly the correction of refractive errors. We must further seek to convince our patient by our methods and manner that we are genuinely concerned in delivering him from his fears, whether real or, as we may style them, imaginary. Only thus can we obtain that true "rapport" which must exist between doctor and patient if success is to be insured. This gained, we may, by simple persuasion, be able to clear away the fear and suffering which may still exist, not because the patient is intentionally exaggerating his symptoms, but because of some unconscious prejudice or habit which is responsible for the trouble. It is useless to treat these cases by stern rebukes or ridicule, however veiled. Action of this kind will only develop resistances which may result in troublesome repression with an intensification of the malady.

Freud points out that the ordinary forgetfulness of every-day life, the slips of tongue and pen and other trivial blunders, are frequently due to an unconscious cause which may be discovered.

Let us take an example from the common experience of a refraction-

ist. The prejudice against glasses is often very great. In the poorer districts of London, and no doubt elsewhere, those who wear glasses are called goggle-eyed or some similar nickname is given to them.

During the war soldiers were greatly prejudiced against glasses, because the army glasses were round instead of oval, as is most commonly supplied in England.

Many girls prefer to endure any amount of suffering rather than wear any form of glasses which may not be regarded as becoming.

The result of this is the development of an unconscious motive, which shows itself in an unwillingness to give a correct history, in mistakes in reading test types, and later on in difficulty with the glasses. These patients have come to us for some real complaint and a desire for relief from their troubles, but when they are brought face to face with the remedy, they shirk the issue. They may be said to regress, to prefer phantasy to reality—in a word, they act like children.

Any one with observation on these lines could multiply instances indefinitely where similar prejudices complicate our practice.

II. PSYCHIC DISORDERS OF A MORE PRONOUNCED FORM ASSOCIATED WITH VISUAL DISTURBANCES

We now come to a second class of cases of a definite psychopathic character, which may vary from the persistent headache to the more pronounced forms of psychic troubles. The nomenclature of these conditions, which includes such terms as hysteria and neurasthenia, is liable to be misleading, so we will not here attempt any definite classification.

It will be sufficient to mention some of the symptoms common to many of these conditions. Headache, minor obsessions or delusions, often visual, tremors of various kinds, disorders of the circulatory, respiratory, or the many glandular systems of the body, may all be met with. Fatigue, inability to concentrate on mental work, varieties of amnesia, insomnia, and phobias of a distressing type may all occur.

Ophthalmologists have for long been aware of the importance of correcting errors of refraction in cases of this kind, but with new knowledge much better results may be anticipated. These are undoubtedly cases which call for comprehensive treatment, and should be dealt with by a psychotherapist, but many of them will first consult an ophthalmologist, owing to the prominence of symptoms referable to the eye and the importance of being able to diagnose the condition is evident.

To take an instance from my own practice:

A patient consulted me on account of symptoms which had shown themselves a few weeks previously on the occasion of the last eclipse of the sun. He had looked at the sun without proper safeguards, and he believed that his eyes had been injured thereby.

He had some conjunctivitis and some slight astigmatism, and both of these conditions were dealt with. There was no sign of any retinal lesion and no scotoma or interference with his color perception. It seemed clear that his main trouble was psychic, and this was confirmed later when, as the result of some slight exposure to glare, he was affected in the same way in spite of wearing Crookes' glasses, and was obliged to give up his work. His medical attendant, a writer on psychologic subjects, formed the same view, and psychotherapy was advised.

This is a common incident in ophthalmic practice, and it is important that it should be recognized, so that it may be appropriately treated. Any attempt to deal with such cases by simply assuring them that there is nothing the matter with them, without recognizing the psychologic aspect, may even intensify the mischief. Here again we see that ophthalmology occupies a central position, with the added responsibility which arises in consequence.

III. PRONOUNCED CASES OF MENTAL DISEASES

In the class of cases which would be certifiable by British law the ophthalmologist is not likely to be concerned except to deal with incidental defects which call for attention. Seeing, however, that the major psychic diseases are usually a development from the minor, it may be possible to prevent the graver forms of disorder by a careful recognition of the premonitory symptoms.

Seeing that visual obsessions are a common element in these cases, it may be of value to inquire into their possible origin.

I was recently asked to see a patient who was in a state of marked dissociation, with visual delusions. She stated that she could see moving objects in the dark corners of the room taking certain definite shapes. The purpose for which I was consulted was on account of defective vision and the possibility of prescribing more suitable glasses. I found that she was developing opacities in the lens which were mainly in the form of minute dots scattered throughout the lens. I am of opinion that these early disturbances of vision due to incipient cataract are quite sufficient to form imperfect images which may grad-

ually materialize into a concept of defined objects, human or otherwise.

Possibilities of the development of serious psychic conditions in connection with cataract, and indeed in other cases of opacities in the media, should be carefully considered, and the experience of ophthalmologists as to the relation between cataract and psychic disorders might be a valuable contribution to psychologic medicine. In a case of cataract which was under my care and operated upon by a colleague of mine the following points may be noted!

Previous to the onset of cataract there were some psychic difficulties, with prejudices and suspicions which were evidently morbid. These were accentuated by other physical infirmities, including deafness, and with the onset of cataract these were much increased. In spite of this mental condition, and indeed with the hope of relieving in some measure these symptoms, and in any case bringing some relief to a condition of pitiable discomfort, it was decided to operate. The change in a monotonous life of removal to a nursing home, with devoted nursing, had a great effect upon the patient. The operation was most successful, and though, as the result of starting up in the night two days after the operation, a prolapse of iris occurred, recovery was satisfactory. During the after-treatment her whole outlook to life seemed to change and the improvement was most gratifying.

Glaucoma again is a disease in which the severity of the pain and the rapid interference with sight are likely to lead to serious forms of phobia. It is worthy of inquiry in these cases as to the possibility of the psychic attitude complicating the physical malady.

These are matters which should engage our serious attention. As ophthalmologists we are brought in contact with disease in all its forms.

As we have already stated, ophthalmology occupies a central position in medicine. Let us prove ourselves worthy of the charge committed to us. It may be that we may be able to give substantial assistance to our colleagues who are grappling with the obscure disorders which afflict the mind, but at the same time we may look for help from them in many of the mysterious ailments which we must deal with in our own sphere.

DISCUSSION

DR. GEORGE F. KEIPER (Lafayette, Ind.): This is an extremely interesting paper because it calls to our attention things which every ophthalmologist ought to know from the standpoint of the new psychology, which starts with

the anatomy and physiology of the central nervous system. We are dealing not with chambers of the mind, but with nerve cells. In the cortex of the brain there are from three billion to eight billion nerve cells, so the physiologists have estimated. These cells are the repositories of impressions, and when they are properly connected up we have that which is called knowledge, and when we take this and go out into the everyday affairs of life and attempt to apply it we get the substance which we call wisdom, and that varies, of course, in different individuals, according to capacity and capability. Of course, we know what some of these nerve cells are for—we have been able to work out what we call localization of function, as along the fissure of Rolando and in the occipital lobes which localize vision, or in the left temporal sphenoidal lobe, where several years ago Dr. Jack, of Boston, and I, simultaneously and independently, pathologically located it by operating for abscess of that lobe, our diagnoses being based upon the symptoms of aphasia which these patients manifested after mastoid operation.

There has been no better definition of memory than that given by Dr. Max Nordau, in his work on "Degeneration," a number of years ago, when he said that memory is nothing more nor less than the product of stored-up nerve-cell action. In the retina of each eye we have the same kind of nerve cells as in the cortices of our brains, and, after all, vision is nothing more nor less than the product of stored-up nerve-cell action. The eye is but a prolongation of the brain in order that the brain may come in contact with that form of motion which we call light and the phenomena produced by it as it comes in contact with all nature.

We have in our offices the same kind of apparatus that the physiologist uses in his laboratory, and we have an exceedingly wide and interesting field for study of psychologic states in relation to these special senses, for, after all, it is through the medium of these seven or eight special senses that we get all our knowledge.

MR. J. GRAY CLEGG (closing): Undoubtedly there are many cases in which we have difficulty in satisfying our patients, the patients who will not take cylinder lenses, who get comfort only when the cylinders are removed. It seems to me some of these are cases where the psychic influence is one of the main factors. Then one occasionally comes across patients who have two sets of glasses from the same prescription—they are perfectly comfortable with one and absolutely uncomfortable with the other, and nothing will persuade them that the two are alike, although the optician and oculist are unable to see the difference. There again we have the psychic element.

In the case of *muscae volitantes* many patients are seriously perturbed in their mind until they are advised not to look at floating spots, and that by suppressing them in their consciousness they will have a return to a state of psychic comfort and rest. I have had two patients, each of whom complained of seeing his nose constantly. The only remedy was either to suppress that consciousness or to have a radical operation undertaken. Undoubtedly in all our work physicians and surgeons are constantly exercising the factor of suggestion to the mind of the patient. Some patients are satisfied with advice in Europe; others come to America to secure their contentment. I am also pleased to be able to say that the reverse holds good.

NUEVO TRATAMIENTO QUIRÚRGICO DEL ESTRABISMO

DR. B. CASTRESANA

Madrid, España

El problema del estrabismo en España es uno de los capítulos de nuestra especialidad que menos ha progresado en la última década del siglo que corremos. La falta de cultura en las masas populares creyendo de buena fé incurable su enfermedad, la resistencia constante para dejarse intervenir quirúrgicamente, el escaso perfeccionamiento de la técnica operatoria y el no poder asegurar al enfermo el éxito seguro y definitivo después de la intervención, son los factores que más poderosamente han contribuido al escaso desarrollo y poca intensidad, que tiene el estudio de la corrección estrábica en nuestra patria.

En España existen multitud de enfermos estrábicos; pero solo se presentan en nuestras clínicas escaso número de ellos, con la pretensión de que centremos bien sus ojos y les demos vista en el ojo que desvían, sin practicarles operación alguna.

Las intervenciones quirúrgicas en los enfermos estrábicos eran escasísimas hace diez años, aún en clínicas numerosas, como la mía. Algunos colegas que me honraban presenciando mis operaciones, al manifestarme deseo de ver operar un estrabismo, tenía que decirles: Los enfermos estrábicos no se dejan operar en España, no por *prejuicio religioso*, como opinaba un ilustre colega extranjero, sino porque creen que su enfermedad no tiene remedio.

En cierta ocasión aconsejaba a una muchacha de diez y ocho se dejase operar un estrabismo interno pronunciadísimo que padecía. Después de exponerle varios razonamientos de índole estética y funcional, cuando creí haberla convencido, me contestó que el *bizquear* los ojos era una *gracia especial* que no estaba dispuesta a corregir. Como se vé, la incultura en el público resistiéndose sistemáticamente al tratamiento operatorio ha sido, es y será en el porvenir, la causa precisamente primordial de que no se haya hecho en nuestra nación un estudio acabado y perfecto de un punto tan importante como es de las desviaciones oculares.

Hay que confesar, sin embargo, que la reacción ha sido tan favorable para nosotros, por el éxito alcanzado con el procedimiento quirúrgico que practicamos en la actualidad, que en estos últimos años el material clínico humano ha sido más abundante, dilatándose mucho el campo de nuestra experimentación.

Los mismos operados de estrabismo, al ver corregida su desviación ocular que no solamente les afeaba, sino que también era en mil circunstancias motivo de burla y de molestia en las distintas manifestaciones de su vida social, han sido los más entusiastas propagandistas, aconsejando a otros individuos que padecían igual defecto se dejen intervenir quirúrgicamente.

La desviación manifiesta de la línea visual en uno de los ojos, o la falta de paralelismo de los ejes visuales, es el defecto ocular que recibe el nombre de estrabismo. Este puede ser paralítico o concomitante, siendo considerado el último como el verdadero estrabismo, porque sin haber parálisis de ningún músculo, no coinciden las dos líneas visuales en el mismo objeto. De otra parte, en esta variedad estrábica, las desviaciones primarias y secundarias con iguales, y no existe la diplopia, porque generalmente la visión imperfecta que el enfermo tiene en uno de sus ojos constantemente, hace que se acostumbre a eliminar la imagen borrosa del ojo estrábico.

El estrabismo concomitante, que a simple vista parece monocular, porque se manifiesta aparentemente por la desviación de uno de los ojos, que es precisamente el que tiene menor agudeza visual, es, sin embargo, bilateral siempre, y el ojo desviado representa, según la opinión de Terrien, la suma de la desviación de ambos ojos. El estrabismo alternante y la igualdad de la desviación primaria y secundaria, nos prueba la existencia de la bilateralidad de la afección, y a la vez nos demuestra que los dos globos oculares están afectados igualmente, sin que exista parálisis muscular de ningún género.

Al comentar este punto del estrabismo el doctor Ribas Valero, uno de nuestros ilustres colegas que mas trabajos ha publicado sobre las desviaciones estrábicas, emite la opinión de Parinaud y dice; "La causa única, tanto del estrabismo convergente como del divergente, es una lesión de convergencia, conservándose íntegra la función de dirección, es decir, una alteración del aparato de la visión binocular, tanto de su parte sensitiva como de su parte motora, y de consiguiente de las conexiones nerviosas que las unen, por donde normalmente se transmiten los reflejos. A estas alteraciones primitivas se agregan posteriormente otras secundarias que las modifican y alteran,

y cambiando su naturaleza producen retracciones fibrotendinosas.” De donde se deduce que para el citado autor francés, el estrabismo consiste en un vicio de desarrollo del aparato de la visión binocular, que afectará a la parte sensitiva y motora a la vez, y que ha de impedir se realice la convergencia de los dos ojos sobre el objeto que miramos. No debe admitirse, por lo tanto, un trastorno muscular orgánico, por que la conservación de los movimientos asociados de dirección, la existencia del estrabismo alternante, la normalidad del campo de mirada cuando se inicia la afección, prueban todo lo contrario.

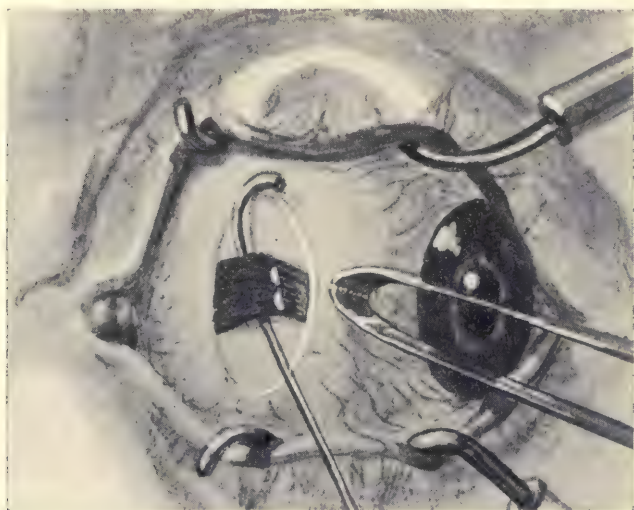


Fig. 1.—Debilitación al músculo antagonista.

El consignar la bilateralidad del estrabismo nos es indispensable si hemos de razonar el tratamiento que empleamos para su curación y nos justifica también nuestra intervención quirúrgica a la vez en los dos ojos, estando en este punto compenetrados con los grandes clínicos Panas y Landolt, que aconsejaban corregir siempre bilateralmente el estrabismo, puesto que el desequilibrio ocular afecta a los dos ojos.

El estrabismo tiene también su tratamiento médico, habiendo sido Javal uno de sus más fervientes partidarios, pero la oclusión de un ojo por largo tiempo, el uso de la atropina, de los vidrios correctores del defecto de refracción, y los ejercicios ortópticos que ocupan un

lugar muy importante con relación al tratamiento quirúrgico en los primeros años de iniciarse el estrabismo, deben sustituirse por la intervención operatoria si no produce su aplicación el efecto apetecido.

Esto no quiere decir que el tratamiento quirúrgico pueda curar por sí solo un estrábico, porque lo único que hace es corregirle su estrabismo aparente, sin intervenir en las funciones de asociación visual, susceptibles de modificar por los medios ópticos que indicabamos anteriormente.

Claro está que no todos los estrabismos son curables, entendiendo por estos aquellos a quienes se pueda devolver la visión binocular, pero

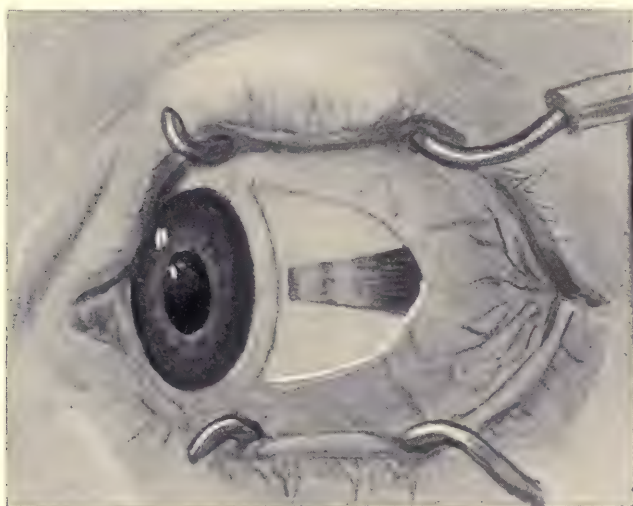


Fig. 2.—Resección de un trozo de la conjuntiva que deja al descubierto la inserción muscular en la esclerótica.

todos se puede decir que son corregibles, es decir, que mediante la intervención quirúrgica podemos suprimirles el estrabismo aparente, y el enfermo queda muy satisfecho desde el momento que le hemos hecho una buena corrección estética, que haga desaparecer su defecto, aún cuando no consiga la visión binocular que nunca tuvo.

Nosotros hemos de procurar la curación verdadera del estrabismo mediante el tratamiento quirúrgico y óptico consecutivo; pero si fuera imposible conseguir la visión binocular, nos conformaremos con haber mejorado la estética facial del enfermo, que tiene capital importancia, sobre todo cuando se trata del sexo femenino.

En el siglo XVIII es cuando parece germinó la idea de poner remedio quirúrgico a la desviación estrábica, relatando Eschenbach, que un inglés llamado Taylor curaba el estrabismo, plegando la conjuntiva con unos hilos de seda, pero no está probado que seccionase el músculo, como opinan algunos autores. La primitiva idea de curación del estrabismo partió de Taylor, pero indudablemente el tratamiento que aplicó fué muy incompleto.

Es preciso que transcurra casi un siglo, hasta el año 1838, para que se hable de nuevo de la operación del estrabismo por Stromeyer, quien hizo una descripción detallada de la técnica operatoria, seccionando el

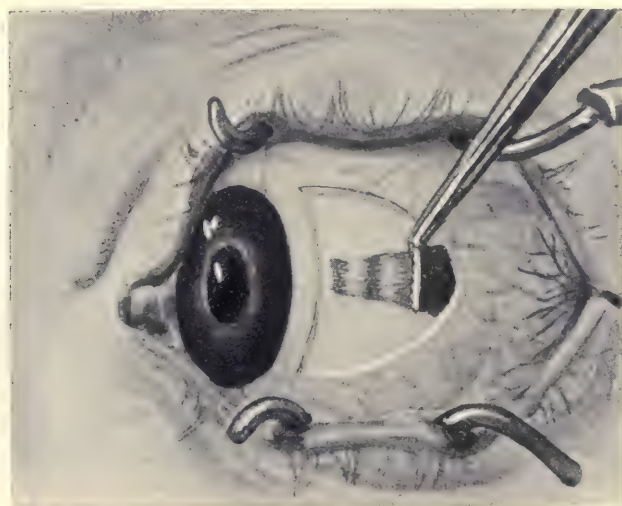


Fig. 3.—Cogida del músculo con la pinza de Prince, a una distancia de su inserción tendinosa, proporcional al grado de estrabismo.

músculo. Pero Stromeyer realizó sus experiencias en el cadáver, siendo Paulín en 1839 el primero que practicó la operación en el ser viviente. Casi al mismo tiempo Dieffenbach seccionaba el músculo en su porción posterior, realizando, por lo tanto, una miotomía, hasta que Guérin en 1840 hizo la tenotomía, que vino a sustituir, aquella, gracias a los trabajos de Bonet de Lion.

Los resultados obtenidos con la miotomía y la tenotomía desanimaron a los operadores. La primera producía un estrabismo paralítico, y la segunda a la larga el estrabismo en sentido contrario. Diez años más tarde, Jules Guérin ideó el avance muscular para con-

trarrestar los estrabismos producidos por la miotomía y tenotomía, mal reglada por aquél entonces.

En los años 1860 al 62 es cuando Graefe, Critchett, Agnew, Cunier, Lucien, Boyer y Bachm, se ocuparon de estudiar con detalle el avance muscular, perfeccionaron la técnica operatoria, y sustituyeron el avance simple por el avance con resección parcial del tendón.

Mas tarde, el año 1883, Wecker ideó el plegado del músculo y practicó el avance capsular, método que aún en la actualidad es seguido por ilustres colegas españoles y extranjeros. En 1890 Parinaud

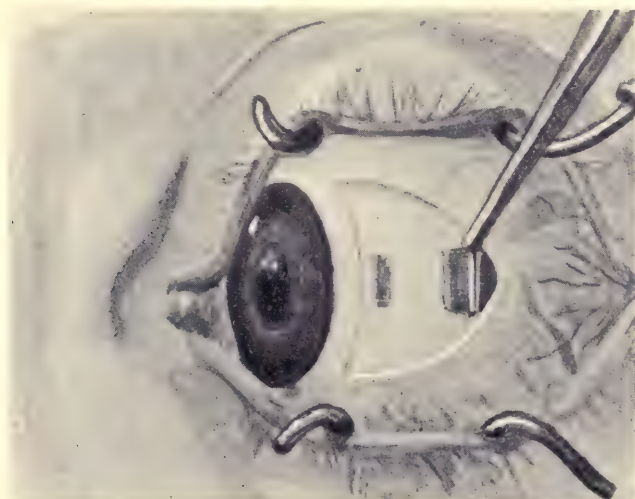


Fig. 4.—El músculo sujeto por la pinza, se secciona por delante de ella quedándose un trozo suelto mayor o menor según la desviación estrabica unido a la esclerotica por su inserción anterior.

sustituye la tenotomía por los desbridamientos capsulares amplios, y Lagleyze propone dos años más tarde el acortamiento muscular. Por último Landolt en 1894 dice que el avance muscular aumenta la energía del músculo y aconseja se practique el avance bilateral.

En realidad, los distintos procedimientos quirúrgicos propuestos para corregir el estrabismo se pueden reunir en dos grandes grupos: En el primero, pueden congregarse los métodos que tienen por objeto disminuir la tracción ejercida por el recto interno, externo, o su cápsula, según que el estrabismo sea convergente o divergente. Entre estos consignaremos la tenotomía y los desbridamientos de Parinaud.

En el segundo, incluiremos aquellas intervenciones quirúrgicas que tienden a aumentar la tracción ejercida por el recto externo y su cápsula, cuando se trata del estrabismo interno, o viceversa, si la desviación estrábica es externa. Este resultado se pretende conseguir mediante el acortamiento y los avanzamientos musculares y capsulares.

Al estudiar cada una de las operaciones propuestas en los dos grupos precedentes, no hemos de exponer su técnica operatoria, descrita en las obras y monografías de nuestra especialidad que tratan

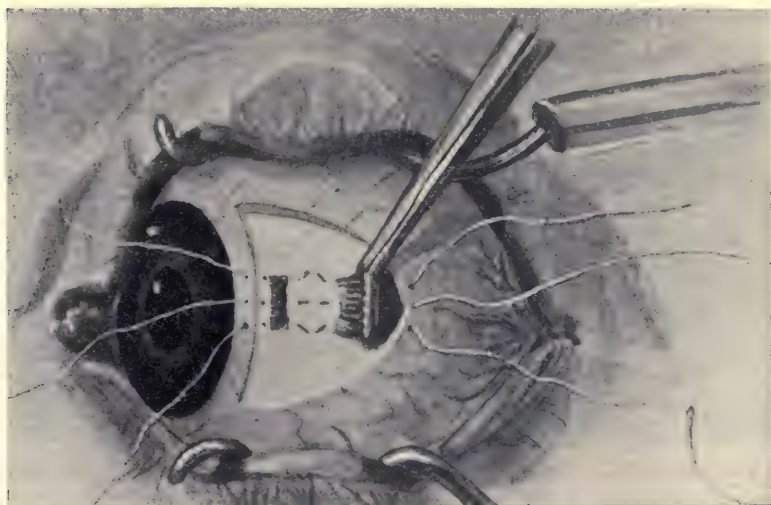


Fig. 5.—Sutura romboidal compuesto de tres puntos, uno central y dos laterales desviados hacia el ecuador del globo ocular y que pueden oblicuarse, si el estrabismo es algo superior o inferior.

del estrabismo: me voy a limitar solamente a exponer sus resultados y consecuencias fisiológicas, después de haber pasado algún tiempo de la intervención.

Modificada y reglada por Bonet de Lion la técnica de la tenotomía, que practicó primero Stromeyer y después Dieffenbach, se puede decir que ha sido la operación que más frecuentemente se ha practicado contra el estrabismo, a pesar de tener, a mi juicio, más inconvenientes que ventajas.

Es indudable que la tenotomía produce, inmediatamente después de practicada, un enderezamiento del globo ocular desviado, pero en

general el enderezamiento no persiste en los días sucesivos, pudiendo algunas veces desaparecer por completo. Sólo en casos muy especiales y en tenotomías muy amplias se ha conseguido alguna vez una corrección de 10 a 12°. Si no hemos suturado la membrana conjuntival, la carúncula lagrimal se hunde, debido a que la conjuntiva del ángulo interno adherida a los tejidos profundos, es arrastrada por el musculo retractado. Se presenta también una exoftalmia postoperatoria que si es bastante acentuada produce una deformidad muy desagradable, que si bien podemos corregir con una cantoplastia del ojo del lado opuesto, no deja de ser una complicación digna de tenerse en cuenta.

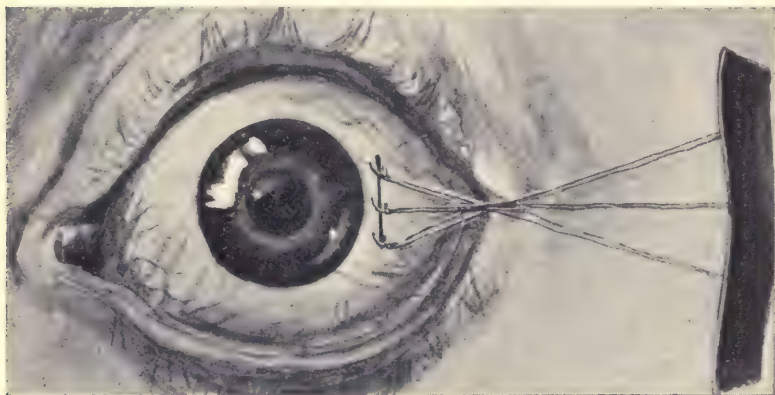


Fig. 6.—Sitio de unión de los puntos de sutura después de verificar el avance indirecto, fijando los extremos de los puntos de seda con un esparandvayso en la parte externa o interna, según el estrabismo que se opere.

Los inconvenientes lejanos de la tenotomía son todavía más graves. Lo más frecuente es que la visión binocular no se restablezca después de la operación, y que sobrevenga el relajamiento espontáneo de la convergencia, dando lugar a un estrabismo externo de un efecto más desagradable que la desviación ocular interna que tenía el enfermo, afectado de estrabismo convergente.

Por otra parte, la tenotomía del recto interno produce un retroceso en la inserción del tendón muscular, que dará origen a una insuficiencia del músculo, y, por consiguiente, de la misma convergencia. No obstante, la tenotomía prudente practicada en el niño que tenga buena agudeza visual del ojo desviado puede en algún caso muy raro determinar el enderezamiento del globo ocular, establecer la visión

binocular y conseguir la verdadera cura del estrabismo; pero estos casos son excepcionales.

Lo que ocurre generalmente es que, si bien al principio el enderezamiento es bastante aceptable, a medida que el niño crece sin que se restablezca la visión binocular, de una parte el relajamiento espontáneo de la convergencia y de otra efecto de la tenotomía, hace que se presente un estrabismo secundario externo que anula por completo la corrección estética que conseguimos en la niñez.

El campo de mirada de los operados de tenotomía se observará que se encuentra disminuido en la abducción del globo ocular, y, en cambio, no se encuentra aumentado en la abducción. Es verdad que el ojo operado presenta un enderezamiento en los movimientos correspondientes a la esfera de acción del músculo tenotomizado, pero es un enderezamiento aparente, realmente débil, al querer seguir a su congénere, no tardando en presentarse la falsa proyección y la desarmonía entre las excursiones del globo ocular.

La visión binocular no se puede, por lo tanto, restablecer, puesto que falta la armonía indispensable en los movimientos de lateralidad y convergencia de los dos ojos. Si la doble imagen generalmente no aparece, es porque existe una ambliopía en el ojo tenotomizado, con neutralización de la imagen percibida. Aunque hayamos conseguido mejorar la agudeza visual del ojo operado, y goce de una visión suficiente, veremos que no es posible se realice la visión binocular, porque entonces aparecerá una diplopia cruzada en el campo de acción del músculo tenotomizado, y como la amplitud de la convergencia se disminuye por haber debilitado el recto interno, la diplopia se presentará en cuanto el enfermo quiera hacer uso de la visión próxima. Por eso dice Landolt que el efecto de la tenotomía separando el músculo recto interno del globo ocular es el mismo que el de una paresia, puesto que dá lugar a una insuficiencia de convergencia, “y, sin embargo, el nombre sol de convergencia decia que no era preciso tomar, sino añadir; no debilitar, sino más bien reforzar.”

El efecto de la tenotomía en el estrabismo divergente, se traduce también por un enderezamiento del ojo desviado; pero como en el estrabismo divergente las desviaciones son muy pronunciadas, el enderezamiento que se consigue tenotomizando es generalmente muy insuficiente. Como persiste la divergencia, cuando el enfermo quiere hacer uso de su visión próxima, se presenta la diplopia cruzada, que será homónima si mira el operado de lejos, pareciéndonos entonces que su bizqueza se interna.

Se comprende que así suceda; porque si el estrabismo divergente resulta de la debilidad de inervación del recto interno, y hemos tenotomizado el recto externo paresiándole, no podrá contraerse lo suficiente en la visión lejana para relajar la convergencia y tendrá necesariamente que presentarse un estrabismo convergente con diplopia homónima, como decíamos anteriormente. Charles Giraud sintetiza su opinión diciendo: "Para tener derecho a tenotomizar el músculo recto externo de un ojo cuya visión es suficiente, haría falta que este ojo presentase un gran exceso de divergencia activa." Onfray se expresa en estos términos: "La tenotomía es una operación peligrosa, cuyo resultado no puede ser absolutamente previsto, y la tenotomía del recto externo es una operación casi siempre inútil y frecuentemente perjudicial." Por último, Parinaud emite su parecer en esta especie de corolario: "A medida que se adquiere experiencia, a medida sobre todo que se persigue en el tratamiento del estrabismo, no solamente el enderezamiento de los ojos, sino el restablecimiento de la visión binocular, tropieza uno con más y más inconvenientes del reculamiento muscular."

Cuenta Paul Charles Giraud, que habiéndose encontrado un día su maestro Rochon Duvigneaud con el célebre anatómico Farabouf le dijo éste en tono sarcástico: "Se cortan todavía los músculos en Oftalmología."

En los Archivos de Oftalmología Hispano-Americanos del mes de Junio pasado, el profesor Landolt, al hablar de los efectos de la tenotomía, dice que produce la desviación del ojo en dirección opuesta, la limitación de la excursión y la falsa proyección en dirección de la acción del músculo. Al tenotomizar los rectos internos en el estrabismo convergente concomitante, se crea una insuficiencia de convergencia que se opone al restablecimiento de la visión binocular, además del efecto antiestético resultante de la retracción de la carúncula. Si por el contrario tenotomizamos los rectos externos en el estrabismo divergente, producirá por igual causa los mismos efectos perjudiciales. Estos hechos, dice Landolt, son la condenación de la tenotomía.

Esta intervención resultaría todavía más perturbadora, a mi juicio, practicándola como intervención única en el ojo que no se desvía, según aconseja Battremieux, el cual corrige indirectamente todo el estrabismo operando sobre el ojo sano, en lugar de limitarse a corregir en este ojo la mitad o poco más, y el resto en el ojo desviado. Este procedimiento produce en un estrabismo convergente, que es la

variedad donde emplea su método, una abducción del ojo fijador igual a la abducción o convergencia estrábica. De este modo, cuando fije en primera posición con el ojo operado, tendrá que efectuar un movimiento igual al grado de desviación corregida, y en virtud de la inervación recuperada por el ojo fijador, para trasladarse desde el sitio donde fué llevado por la tenotomía, hasta colocarse en primera posición, el ojo desviado que permanecía indiferente en su posición anormal, se enderezará por sinergia hasta que alcance la primera posición. Este procedimiento no ha sido practicado mas que por su autor.

De otra parte, el efecto corrector de la tenotomía no se interpreta de igual modo por los diversos autores que se han ocupado de esta cuestión. Las dos hipótesis que más partidarios han tenido, son la de Parinaud y la de Motais. Para el primero, la tenotomía remedia un trastorno de inervación y obra debilitando el músculo que retrocede de su inserción. Esta debilidad, esta insuficiencia muscular, se podría explicar por la disminución de extensión, a consecuencia del enroscamiento del músculo. En cambio, el segundo fundamente su opinión en el papel importantísimo que juegan las aletas ligamentosas de la cápsula Tenon, que se prolongan hasta el borde orbitario correspondiente donde tienen su inserción.

Estas aletas, supone el profesor Motais, que limitan la contracción de los músculos, porque son susceptibles de alargarse en cierta medida cuando practicamos la tenotomía. En virtud de este alargamiento, la aleta dispondrá, por consiguiente, de una porción menor susceptible a ceder en la contracción muscular. El músculo tendrá que luchar desde el principio de su contracción, con una tensión mayor de la aleta ligamentosa, fácilmente comprensible, por haber retrocedido el tendón, al separarse de la membrana esclerotical. Esto nos explicaria la disminución en la excursión del ojo, y la debilidad o insuficiencia del músculo tenotomizado.

El Dr. Lagleyze no acepta la hipótesis de Motais, porque fundamenta sus experiencias en disección de animales muertos, es decir, en las peores condiciones para resolver un problema de dinámica muscular, y además, porque dice que el dibujo presentado por Motais para sus explicaciones no coincide con los casos, porque la figura debería representar en iguales condiciones estáticas de sus músculos, la posición del estrabismo y la de su corrección, para observar el estado del músculo y de su aleta ligamentosa, antes y después de la operación.

No hemos nosotros de seguir las disposiciones científicas que hace

tan sabio profesor con respecto a la interpretación de los esquemas indicados, puesto que no es de nuestra incumbencia en este momento; sólo daremos a conocer sintéticamente su opinión, que al fin y al cabo, es una hipótesis más en el campo de la ciencia.

En la tenotomía dice: "Lo primero que se produce es un enderezamiento del ojo. El músculo antagonista libre de la contractura del músculo tenotomizado, disminuye su longitud, por su propia conicidad. Abierta la cápsula de Tenón al practicar la intervención, permite que la elasticidad propia de la hoja periorbitaria de esta cápsula, se retracte sobre el borde orbitario correspondiente al lado operado, y el globo del ojo, una vez disminuidas las adherencias que le detenían en el sitio del tendón cortado, se dirige hacia adelante, volviendo al mismo tiempo hacia el lado opuesto." El resultado, por lo tanto, después de la tenotomía, será que el músculo conserva sus dimensiones anteriores, obedece como antes al mismo influjo nervioso, no está debilitado, y tiene la misma contractilidad, o mayor que antes de la operación. En la tenotomía de un músculo el que se acorta es el antagonista, en una cantidad proporcional correspondiente a la corrección del estrabismo, quedando el músculo tenotomizado con la misma longitud que antes de la intervención.

En vista de los inconvenientes que tiene la tenotomía, se le ha querido sustituir por otros procedimientos quirúrgicos, que respetando la inserción anterior del músculo no la debilite. Empecemos por los desbridamientos capsulares de Parinaud.

En el año 1890, Parinaud ideó su operación de los desbridamientos, que vino practicando hasta su muerte, como verdadero sustituto de la tenotomía. El ilustre oculista fundamentaba su intervención quirúrgica, en que la extensibilidad del músculo está sujeta por sus conexiones con la cápsula, cuyas suspensiones fibrosas podrían en los estrabismos antiguos presentar adherencias patológicas, que es preciso liberar para el enderezamiento del globo ocular.

El desbridamiento capsular de un músculo ha de ir acompañado siempre del avance del antagonista resultando la operación combinada, puesto que se interviene en los demás músculos a la vez.

Según el Dr. Parinaud, la operación combinada producirá un enderezamiento inmediato de 25° a 30° que yo no he podido comprobar. Es preciso tengamos una asepsia rigurosa y no traumaticemos mucho en la operación, porque nos puede venir una retracción inflamatoria secundaria, que neutralice por completo el efecto, que en un principio se consiguió con el acto quirúrgico. Este procedimiento

tiene la ventaja de que no modifica la movilidad del globo ocular hacia su parte interna, y por lo tanto, no se opone al restablecimiento de la visión binocular, porque no hace más que librar al músculo de sus adherencias respetando su inserción anterior.

Sólo a título de curiosidad merece consignarse el alargamiento muscular de Landolt, puesto en vigor el año 1905. En este procedimiento como lo indica su nombre, el objeto es alargar el músculo encogido, o mejor dicho acortarle, respetando su inserción anterior.

En ocasiones, en vez de alargar el músculo se le secciona, y entonces nos encontramos con los graves inconvenientes de la miotomía de Dieffenbach, y así lo comprendió el mismo Landolt al limitar mucho sus indicaciones.

Julio G. Guérin, en 1849, dió a conocer el nuevo procedimiento quirúrgico del avance muscular, con el que pretendía corregir el estrabismo secundario consecutivo a tenotomías desgraciadas. En 1872 Critchett modificó su técnica empleándole como tratamiento de las desviaciones oculares primitivas, y Wecker en 1883, en vez de separar el músculo de su inserción tendinosa, lo plegaba sencillamente, practicando un avanzamiento cápsulomuscular, asociado casi siempre a la tenotomía del músculo antagonista, que fué suprimida más tarde por Landolt.

Este colega, partidario entusiasta de los avanzamientos, desecha las tenotomías y no se contenta sólo con el aislamiento del tendón, sino que reseca la parte anterior del músculo, para llevarle más cerca del limbo esclerocorneal, constituyendo la operación del avanzamiento con resección muscular.

El procedimiento de Wecker se caracteriza por la plegadura del músculo y de su cápsula, respetando la inserción tendinosa de los rectos. El efecto correctivo que conseguimos con esta operación varía según la mayor o menor extensión del plegamiento y según la combinemos o nó con la tenotomía. Si practicamos el procedimiento clásico de Wecker, avanzamiento con tenotomía del antagonista, la desviación estrábica se corregirá de 15 a 20°, porque el avance capsular aumenta la acción correctora de la separación muscular, pero en cambio disminuye la abducción y la amplitud de la convergencia, factores que más tarde vendrán a perturbar la visión binocular.

Si hacemos caso omiso de la tenotomía, e intentamos corregir el estrabismo empleando el avance capsular bilateral, observaremos que no corrige más de 10 a 12°, pero no perturba la convergencia, y puede producir la curación verdadera de estrabismos muy poco pronun-

ados. La corrección que produce el avance capsular bilateral es, por lo tanto, muy limitada y se disminuye, porque al quitar los puntos de sutura desaparece la plegadura del músculo. La cápsula tenoniana no queda avanzada según parecen demostrar las operaciones hechas por Halt en los animales. Es posible, dice Parinaud, que los hilos de sutura determinen en el músculo y la cápsula una ligera retracción cicatricial. En suma, el inconveniente del avance cápsulomuscular es que no corrige más que las desviaciones estrábicas muy débiles. En mi humilde opinión, de iguales defectos adolece el acortamiento muscular de Lagleyze aún asociado a la tenotomía. Sólo corrige las desviaciones estrábicas pequeñas, si no se practican amplias tenotomías con todas sus consecuencias. Los estrabismos pronunciados aunque parecen corregirse después de la intervención, vuelven a reaparecer de nuevo con igual intensidad al cabo de cierto tiempo.

La verdadera intervención quirúrgica que merece el nombre de avanzamiento muscular, se realiza cuando separamos el músculo de su inserción y lo trasplantamos más cerca del limbo esclerocorneal. Las modificaciones que se han hecho en la técnica clásica del avanzamiento por Jocqs, no resecando la conjuntiva; el avanzamiento en A de Valude; el avanzamiento y sus cápsulas en sutura por Rochon Duvigneaud, y la sustitución de los hilos de seda por tendones de renguifo, para suturar la esclerótica y no tener que quitar los puntos de sutura, empleados por Terson, han dado casi los mismos resultados. El procedimiento del avance musculocapsular con sutura especial de Jocqs, presentado en el Congreso de 1903, y los distintos trabajos de este ilustre publicista, dados a conocer los años 1904 y 1909 en *La Clinique Ophtalmologique*, hicieron que se emplease su técnica operatoria por algunos oculistas con resultados halagueños en los estrabismos débiles. En los pronunciados, aconseja Jocqs que se practique una pequeña tenotomía del músculo antagonista, por resultar poco enérgico su procedimiento.

En el avance muscular admite Motais el aumento de la fuerza y la extensión del músculo avanzado, fundándose en la hipótesis de que la aleta ligamentosa del músculo que se avanza, se relaja. Para demostrar su opinión presenta unos dibujos artificiosos que según Lagleyze tienen el mismo error señalado en los esquemas de la tenotomía. La aleta ligamentosa antes y después del avance muscular, ocupa el mismo sitio en el espacio orbitario, dice Lagleyze, y, por consiguiente, las aletas no pueden influir aumentando la acción del músculo. Este no avanza en el espacio orbitario como parece des-

prenderse de las figuras de Motais, porque sería preciso para ello que el globo ocular se mantuviera fijo y no estuviera suspendido en la cavidad orbitaria, como una especie de articulación del género de las enartrosis.

Para el profesor de la Universidad de Buenos Aires el músculo no aumentará la fuerza, por crearse un nuevo obstáculo en el aumento del antagonista, que ha de neutralizar, empleando parte de su energía, lo que no ocurría antes de la intervención, puesto que el antagonista en posición estrábica tenía un estado de menor tensión. Si nosotros no aumentamos la fuerza de un músculo debilitado, con el avance del mismo, creo que podemos por lo menos aumentar la eficacia de su contracción, como afirma Landolt en su reciente trabajo, publicado en los *Archivos de Oftalmología Hispano-Americanos* en el mes de Junio del año anterior. Al avanzar el músculo, aumentamos la extensión de contacto sobre el globo ocular, lo que producirá un efecto suficiente, a pesar de su fuerza escasa.

El avance muscular origina por sí solo un enderezamiento del globo ocular, sin producir exoftalmía ni deformación de la hendidura palpebral. No disminuye la abducción, y la amplitud de la abducción se ha conservado. El estrabismo secundario no se presenta en el transcurso del tiempo, y con ejercicios constantes de visión binocular, pueden algunos casos de estrabismo muy poco pronunciado llegar a la verdadera curación. El inconveniente de este procedimiento es que no produce más que un enderezamiento insuficiente, no sirviendo para los estrabismos de un grado elevado.

Para las desviaciones estrábicas muy pronunciadas Landolt recomienda las resecciones parciales combinadas con el avance, método en realidad que se diferencia muy poco en su técnica del avance muscular sencillo.

El enderezamiento que se consigue es, sin embargo, mejor, y aumenta tanto más cuanto mayor sea la resección muscular que practiquemos, sin que se observe estrabismo secundario. La corrección es más acentuada, y, con el empleo de los medios ópticos y ortópticos, se consigue en algunos casos la verdadera curación. Esta se comprende, si tenemos en cuenta que el músculo, después de la intervención quirúrgica, no solo queda arrollado sobre el globo ocular, sino acortado, lo que acrecienta su acción, sin perjudicar el funcionamiento del antagonista, para que pueda realizarse la visión binocular. Desgraciadamente, la corrección no suele ser duradera en la mayor parte de los casos, cuando el avance con resección ha sido monocular,

pues bien pronto vuelve a presentar de nuevo el desequilibrio muscular, cuando nuestra intervención se limita sólo a un ojo.

Es verdad que aparentemente, siendo solo uno el ojo desviado, parecer natural que la operación no debe realizarse más que sobre el globo ocular que se desvía; pero la observación demuestra que la unilateralidad del estrabismo no existe más que de una manera aparente, y que la bilateralidad, cuando no es espontánea podemos provocarla mandando al enfermo cubrir el ojo que habitualmente fija, lo que dará origen a la alternativa de la desviación igual para los dos ojos. El estrabismo al parecer monocular, dice Onfray, "se debe a que uno de los ojos está forzosamente dirigido hacia el objeto que el individuo quiere ver; pero en realidad se trata de un espasmo de la convergencia, función eminentemente binocular por su misma definición. Es, pues, lógico repartir el efecto operatorio sobre los dos ojos, y los resultados han demostrado que este método, no rompiendo el equilibrio normal binocular en los movimientos asociados, facilita la verdadera cura."

Algunos ilustres colegas sostienen nuestra misma opinión, y creen que siendo una afección binocular se la debe aplicar un tratamiento quirúrgico bilateral. Consecuente con este principio, Pannas practicaba la doble tenotomía de los rectos internos y externos, según fuera el estrabismo convergente o divergente, y Landolt, el doble avanzamiento de los rectos externos o internos, según se trate de una desviación estrábica interna o esterna.

Si echamos una rápida ojeada sobre las opiniones emitidas por diversos autores, veremos que casi todos hablan de la tenotomía, del avance cápsulomuscular, de avance muscular unilateral con resección, de la tenotomía combinada con el avance; pero ninguno menciona el avance bilateral indicado por Landolt. Es más, asún después de haber sido preconizado por tan eminente colega, apenas si lo han practicado aquellos oculistas que más estrabismos han tratado quirúrgicamente.

En efecto; si recorremos el capítulo del estrabismo en algunas publicaciones modernas, no encontramos en ellas la recomendación del doble avance muscular puro y simple, hecha por Landolt.

En el trabajo de Guedel (1875) sobre la patogenia y tratamiento del estrabismo después de emitir la idea de que el verdadero tratamiento del estrabismo es el quirúrgico, dice que debemos practicar la tenotomía sola o combinada con el avance del músculo antagonista, verdadera panacea de la corrección estrábica.

Testut, en 1881, al publicar el avanzamiento del tendón en el tratamiento del estrabismo, manifiesta que hace falta repartir entre los dos ojos la corrección de la desviación, pero asociada siempre a la tenotomía del recto interno.

En la publicación de Bonnemaison el año 1882 acerca de los diferentes procedimientos quirúrgicos para el estrabismo monolateral excesivo, también indica que debe practicarse a la vez el avanzamiento del recto externo y la tenotomía. El mismo consejo de Lagrave en 1893 en su trabajo de contribución al estudio del tratamiento quirúrgico del estrabismo, afirmando que la base del tratamiento radica precisamente en la tenotomía.

Más tarde, tres años después, en 1896, Langle en nada modifica las opiniones anteriores, hasta que en 1906 Alberto Delbarne en su "Estudio clínico sobre el tratamiento operatorio del estrabismo concomitante interno," indica que en los casos de gran desviación debe intervenir en los dos ojos, practicando el doble avance, pero asociado siempre a la tenotomía, como venían preconizando los demás autores. Wecker discute sobre las tenotomías simples, o las tenotomías con avanzamiento, y Fuchs y Kavel no hablan del doble avance, el primero en su Tratado, y el segundo en su Manual de estrabismo. Lo mismo le sucede a Bielchowsky en el Tratado reciente de Axenfeld del año 1914. Lagleyze en su Tratado del estrabismo, año 1913, aconseja su acortamiento muscular especial, sin separar el músculo de la inserción, con o sin tenotomía del antagonista.

En el Congreso de la Sociedad Francesa de Oftalmología de 1893, Parinaud, que era el penente para el tratamiento del estrabismo, ni siquiera menciona el doble avanzamiento muscular. Morax y Terrien se limitan en sus modernos Tratados, a citar sencillamente la operación. Solamente Landolt vuelve a insistir, después de haber emitido la idea del doble avance, en la necesidad de que la operación se generalice, siendo Benet Onfray el que más la ha dado a conocer en su magnífico trabajo sobre el estrabismo, publicado en 1907.

Antes de esta época, en 1905, Rochon Duvigneaud había empezado a tratar a los estrábicos por doble avanzamiento de los rectos externos con resultado positivo. Los éxitos que consiguió, le hicieron aplicar este método a la mayoría de los estrábicos que operó en los Hospitales de Laënnec y de la fundación Rothschild. Su discípulo predilecto, Giraud, en su notable trabajo sobre el doble avanzamiento muscular, al hablar de su maestro, dice: "El no cree que toda la desviación pueda ser corregida por el doble avance más considerable posible, es decir,

con resección importante del tendón muscular," pero le considera un procedimiento excelente.

El Dr. Giraud, consecuente con la doctrina de su maestro, cree que no se puede desterrar la tenotomía actualmente en el tratamiento del estrabismo si bien ha de reservarse sólo para ciertos casos, a causa de los inconvenientes que tiene para que se realice la convergencia, debiendo procurar obtener la mayor ventaja con el doble avanzamiento muscular.

En cambio, hay otros oculistas como Parinaud, que solamente cuando el resultado obtenido por la intervención unilateral resulta incompleto, es cuando interviene en el ojo fijador, Lapersonne en las desviaciones débiles tampoco interviene mas que en un ojo, y es preciso que la desviación pase de más de 10° para que practique la intervención quirúrgica bilateralmente. Por mi parte, de conformidad con la opinión de Wecker, creo que la intervención unilateral podría aplicarse en alguno de los casos de estrabismo que él llama incorrigibles, en los que no podemos esperar más que una corrección de su deformidad, pero nunca en los curables, que son susceptibles de recuperar la visión. En éstos, la intervención a mi juicio debe ser siempre bilateral, respetando al sinergia binocular.

La opinión de Rochon Duvigneaud, cuando dice: "En aquellos casos en que los músculos rectos externos muy débiles se oponen a los rectos internos muy fuertes, el doble avanzamiento muscular por extenso que sea resulta insuficiente, es cierto;" para que el avance dé resultado, tendremos que debilitar los internos. Cómo realizaremos esta debilitación? Para Rochon Duvigneaud, por un procedimiento ignorado aún, porque la tenotomía es una operación brutal, excesiva e imposible de dosificar.

En efecto, esta operación que todavía la practican algunos oculistas y que en algunos casos muy excepcionales puede llenar alguna indicación, podría tener su apogeo antiguamente, cuando se admitía la teoría muscular del estrabismo, pero en la actualidad no debe practicarse *larga manu*, como se hace, porque al debilitarse el músculo, no sólo se disminuye la abducción y la amplitud de convergencia, que son factores indispensables para restablecer la visión binocular, sino que produce alguna vez el estrabismo secundario.

¿Se debe abandonar en absoluto la tenotomía? Creo que la tenotomía bilateral no debe practicarse nunca; únicamente la de un solo lado puedo estar justificada, cuando se trata de un estrabismo con ambliopía acentuada, cuando la contractura muy marcada del mús-

culo se acompaña de cambios de estructura con pérdida de su elasticidad, y cuando el enfermo es de edad avanzado, y no dispone del tiempo necesario para someterse después de la operación a los ejercicios ortópticos que le pudieran reintegrar a la visión binocular. Aún reuniéndose todas estas circunstancias, se debe hacer la tenotomía con gran prudencia y sólo en las desviaciones estrábicas muy débiles, puesto que hemos indicado que la tenotomía produce una paresia incurable, que dificulta convergencia.

Si la asociamos al avanzamiento, constituye la operación combinada, que produce un enderezamiento del globo ocular mucho más acentuado. Este hecho no se puede negar, porque la clínica nos lo confirma algunas veces; pero, de admitirla, a creer, como dicen los partidarios de la tenotomía combinada, que son menos marcados sus inconvenientes, porque la debilitación del músculo es limitada por el obstáculo que opone el avance del antagonista a la exoftalmía, hay una gran diferencia. El resultado que se obtiene con algunas tenotomías combinadas, depende, como dice Onfray, del músculo que hemos avanzado en modo alguno del que hayamos tenotomizado.

No puedo tampoco admitir la opinión de Worth, al suponer que en los grados considerables el estrabismo, no se puede enderezar el globo ocular con los avances sin producir una enoftalmía, porque tengo operados de estrabismos muy fuertes con el doble avanzamiento indirecto, sin que se haya presentado el fantasma enoftálmico. Debo confesar que en estos enfermos no se practicó el doble avance sencillo, lo asociamos a la debilitación del músculo antagonista, en la forma que indicaré al hablar de la técnica operatoria que ejecuto generalmente, cuando opero estrabismos pronunciados. Los inconvenientes de la tenotomía no se evitan ni disminuyen en lo que se relaciona con la convergencia asociándola al avanzamiento, y sólo deberá practicarse en algunos casos muy especiales como indicaba anteriormente.

La operación que no tiene ninguno de los inconvenientes de la tenotomía es el avance muscular, que yo llamo indirecto, que no solamente nos corregirá el estrabismo, sino que muchas veces devolverá al enfermo estrábico la visión binocular perdida. La objeción que se ha hecho a los avances diciendo que tiene unos efectos correctores limitados, puede tener algún valor, cuando se trate el avance capsular, o muscular unilateral, que es insuficiente, pero la citada objeción no puede hacerse cuando se practique el avance bilateral, con resección muscular que duplica el efecto de la operación.

En el caso de una desviación muy débil, en la que no podamos ob-

tener la curación verdadera, por alteraciones irremediables en el aparato dióptrico, el avance muscular unilateral puede ser algunas veces suficiente; pero si queremos baucar la visión binocular, la corrección debe ser repartida entre los dos ojos. La aplicación de distintos avances para cada variedad de desviación no la podemos admitir en la actualidad. Es inadmisibile establecer, que para un estrabismo inferior a 10° , se practique un avance capsular, para uno de 15 a 20 muscular, y si pasa de 25, se refuerce con la resección del músculo.

Al radicar la desviación estrábica, al parecer, sobre un ojo solo, lo natural es que la operación recayese solamente sobre el ojo desviado pero la unilateralidad del estrabismo, repito que es aparente, la desviación estrábica es alterante, lo que se comprueba mandando tapar al enfermo el ojo que fija, como dejo indicado; pues entonces veremos que hay desviaciones iguales en los dos ojos. Si la desviación, por lo tanto, es bilateral, el efecto operatorio deberá repartirse entre los dos ojos, puesto que ambos se hallan afectados.

El doble avance muscular indirecto, por acortamiento de los músculos, lo practicaremos en el estrabismo convergente, avanzando los dos rectos externos indirectamente, como describirá después, y en el divergente los rectos internos. Este avance no disminuye el campo de adducción, aumenta el de abducción y respeta la convergencia. Debe practicarse siempre bilateralmente, si perseguimos conseguir la visión binocular del enfermo. Con el avanzamiento muscular doble indirecto por resección del tendón, que le injertaremos después de acortado en su primitivo punto de inserción, aumentamos de una manera considerable la potencia de enderezamiento del avance muscular, y conservaremos siempre la convergencia. Las excursiones nasales no pierden nada, las temporales se normalizan, y los ojos se mueven correctamente. Muchas veces se obtiene la visión binocular, y una amplitud de convergencia normal.

Al ocuparse el Dr. Rochon Duvigneaud del doble avanzamiento, no pretende corregir todas las desviaciones por esta operación. Cuando los rectos internos son muy fuertes en el estrabismo convergente, y los rectos externos muy débiles, dice que será preciso además de los avances, disminuir la energía potencial de los músculos rectos internos, por algún medio especial, que quizás pudiese ser seccionado alguna fibra muscular en todo el ancho del músculo, porque la tenotomía es una operación que no se puede dosificar.

En el estrabismo divergente lo mismo que el convergente, la acción

correctora del doble avance indirecto es buena y podemos aumentarla resecaando una porción mayor o menor del músculo, según nos convenga.

La abducción no disminuye con la operación y en cambio conseguimos que aumente notablemente la adducción. En esta variedad de estrabismo el campo de adducción está limitado en los dos ojos y mejora con el avance bilateral.

A los rectos internos les proporciona una contracción suficiente y paralela para poder realizar la visión binocular, sobre todo en aquellos estrábitos que tienen la facultad de fusión muy desarrollada. Con los avances, por lo tanto, conseguiremos una corrección estética y funcional a la vez, que es precisamente nuestro bello ideal.

Claro está que no se pueden corregir los estrabismos muy pronunciados con solo el avance bilateral indirecto, pero si lo asociamos a la debilitación del músculo antagonista, en la forma que indicaré, el problema de la corrección del estrabismo queda resuelto para todos los casos que se nos puedan presentar.

La técnica operatoria que practicamos en el avance bilateral indirecto con debilitación del músculo antagonista, se diferencia de la que emplean otros eminentes colegas, en detalles, tan importantes, que la podemos considerar como nueva, aunque se nos diga parodiando a Salomón que *nihil novum sub sole*. Es cierto que nada puede considerarse como nuevo, pero si a un procedimiento operatorio se le modifica en su principio fundamental, y además se le añade un acto quirúrgico que viene a complementarle, y corregir las deficiencias que tiene, creo nos podemos considerar autorizados para calificarle como nuevo.

¿Cómo se debe realizar el procedimiento operatorio ideal del estrabismo pronunciado? En la forma que vamos a describir:

Se hace anestesia local mediante inyecciones subconjuntivales y musculares con solución en suero fisiológico de novocaína y adrenalina que es suficiente, o anestesia general con cloroformo que es la que prefieren casi todos los enfermos para evitar el dolor por completo. Anestesiado el paciente en cualquiera de las dos formas, se desinfecta el campo operatorio, por medio de repetidos lavados con la solución acuosa al 4 por 100 de ácido bórico, y se empieza la operación debilitando los músculos rectos internos en el estrabismo convergente, para después practicar el doble avanzamiento indirecto, o viceversa, se debilitan los externos y se avanzan los internos cuando se trata de un estrabismo divergente.

Para debilitar el músculo que es el punto más original e importante de mi intervención, se empieza por aislar su tendón en la forma que lo hacemos cuando se desea separar lo del globo ocular. Colocado el separador palpebral, se coje con la pinza la conjuntiva a unos cuantos milímetros del limbo esclerocorneal. El pliegue que hemos levantado de la membrana conjuntival se escinde de un tijeretazo, y nos queda de este modo abierto en ojal en la conjuntiva, de unos cinco o seis milímetros de extensión aproximadamente.

Después, con ayuda de las tijeras disecamos los labios del ojal, levantando la conjuntiva en todas direcciones hasta poner al descubierto la inserción tendinosa del músculo, que estará cubierta por la cápsula. Cogemos a esta con la pinza al nivel del borde superior del tendón, y con la punta de la tijera la incidimos produciendo una pequeña abertura por donde se introduce el gancho de estrabismo. La punta de este instrumento dirigida hacia atrás rasando sobre la esclerótica pasa por debajo del músculo, y se presenta cubierta por la cápsula al nivel del borde inferior del tendón muscular.

Se dá un corte con la tijera en la cápsula y queda libre la punta del gancho. Se introduce otro por la abertura practicada, que sustituye al primero y que camina en sentido opuesto, con el fin de coger el tendón en su totalidad, y una vez conseguido, se levanta con fuerza sujetándolo con la mano izquierda. Colocado el tendón en esta forma, con la tijera o un bisturí, se dan dos cortes en dirección vertical, a unos dos o tres milímetros por detrás de la inserción que tiene el músculo con la esclerótica y en una extensión variable, según queramos debilitar mas o menos la potencia muscular. Los citados cortes serán practicados en las partes intermedias, sin cortar nunca las fibras correspondientes a los bordes musculares, superior e inferior, ni tampoco las correspondientes a la parte central, con el fin de que no se modifique el centro de rotación del globo ocular. Cortada en esta forma la cantidad del músculo que nos convenga, se saca el gancho que lo sujetaba y se reunen los bordes cruentos de la conjuntiva, mediante un punto de sutura, pasando inmediatamente después a practicar el avance bilateral indirecto de los músculos debilitados.

Se coloca el blefarostato y se desinfecta el ojo en igual forma que lo hicimos para la debilitación del músculo. Después un ayudante por medio de una pinza para fijar, colocada cerca del limbo, sostiene por dentro del globo ocular, mientras que el operador coge con la

pinza un pliegue de la conjuntiva, a unos cuatro milímetros del limbo esclerocorneal y de un tijeretazo lo escinde, reseca una pequeña porción de conjuntiva en forma de media luna de concavidad corneal, que deja al descubierto la inserción muscular en la esclerótica. Descubierta la inserción del tendón, se coge la cápsula en el borde superior del músculo, y con la punta de la tijera se practica en ella una pequeña abertura, por la que se introduce un gancho fino de estrabismo, que llevándolo al ras de la esclerótica por debajo del músculo, viene a levantar en su borde inferior la cápsula que se desprende de un tijeretazo.

Cogido el tendón muscular en toda su extensión con el gancho, se disea el músculo con tijeras curvas de punta roma, aislándole de la conjuntiva de la cápsula y de tejido episcleral. Separado completamente, se levanta con la ayuda del gancho de estrabismo, para sujetarle con la pinza de Prince, a una distancia de su inserción tendinosa proporcional al grado de estrabismo. Colocado el músculo en esta situación, se secciona al ras de la pinza, por delante de ella, quedándonos un trozo suelto de músculo mayor o menor unido a la esclerótica por su inserción anterior, que será el que resequemos después de colocar la sutura en *rombo* que describiremos más tarde.

El trozo de músculo que resecamos, es más o menos extenso, según sea mayor o menor la desviación estrábica, calculando su extensión con bastante exactitud en la actualidad, a consecuencia de los estudios comparativos que hemos hecho en la clínica al ver los efectos que producen las distintas cantidades de músculo resecado.

Desde luego podemos decir que no se pueden dar dosis de resección con exactitud matemática, por cada grado de desviación, pero podemos asegurar, que no se deben tener las amplias resecciones, y menos si se aplica este calificativo a la resección de 4 ó 5 milímetros como quiere Landolt. En los estrabismos muy pronunciados he llegado yo a resecar hasta 12 y 15 milímetros con un resultado muy satisfactorio.

El Dr. Jenaro González, uno de los oculistas españoles que ha estudiado con más entusiasmo el problema quirúrgico del estrabismo, en su comunicación a la Academia Quirúrgica el año 1915, acerca de modificaciones quirúrgicas al procedimiento operatorio de resección tendinosa Schweigger, establece un cálculo matemático para precisar con exactitud la cantidad de tendón que hemos de resecar, conforme el ángulo de desviación estrábica. Según sus cálculos matemáticos, la cantidad de tendón que podemos resecar es de 2 milímetros por cada 10° de desviación. En la clínica esta cantidad resulta algo

insuficiente, y el resultado de su modificación al procedimiento Schweigger no lo cree muy seguro cuando dice: "En algunos casos puede completarse la operación alongando el músculo antagonista, según el método de Verhoeff."

Es difícil responder a esta cuestión con una cifra exacta, dice Lagleyze, al hablar del acortamiento muscular, porque la solución depende de causas desconocidas que pueden cambiar según las circunstancias. Entre ellas las resistencias de los diversos tejidos, que varían de un sujeto a otro, en virtud de la edad, de la antigüedad del estrabismo, de las diferencias de la estructura de los músculos, o en las adherencias anormales que han podido realizarse en el transcurso del tiempo, del conocimiento exacto de la resultante de fuerzas, que acentúan sobre el ojo en distinta dirección, al ejecutar sus movimientos, y de causas funcionales o anatómicas imposibles de resolver. De este modo, puede explicarse como en dos estrabismos de un grado igual de desviación, una misma cantidad de músculo resecado puede dar un resultado diferente. No obstante, creo se puede calcular aproximadamente una resección muscular de dos y medio milímetros por cada diez grados, por lo que he podido observar en la clínica.

El mismo Dr. Gonzalez expresa, que la solución matemática aplicable a los tejidos tiene su límite, porque en ellos intervienen ciertos factores que no es posible solucionar con números. Los músculos, dice Onfray, no son cintas inertes, sino tejidos que tienen una contractilidad propia, sometida a la acción más o menos intensa de la energía nerviosa, y aunque pudiéramos calcular con exactitud matemática la mudanza de inserción del músculo resecado, aún en este caso, nos sería imposible precisar exactamente la cantidad de tendón que debemos resecar, porque la práctica clínica demuestra que tal precisión es imposible.

El trozo de tendón libre, separado de la pinza de Prince al ser cortado el músculo que permanece unido a la esclerótica en su parte anterior, nos sirve de punto de apoyo para sujetar con la pinza el globo ocular, al colocar la sutura en rombo, que es otro de los puntos diferenciales importantes de este procedimiento.

La sutura en rombo la componen tres puntos, uno central y dos laterales. El primero sigue el diámetro horizontal. Comienza penetrando desde cerca de la cornea por debajo de la conjuntiva y de algunas fibras escleróticas, pasa por debajo de la inserción del tendón por donde sale, penetra de nuevo en el espesor de las fibras de la esclerótica por donde camina en una extensión de unos dos mili-

metros, y finalmente, pasando de dentro a fuera por detrás de la pinza de Prince, el músculo, la cápsula y la conjuntiva, viene a salir cerca del ángulo palpebral externo.

Los otros dos puntos laterales de la sutura superior e inferior, empiezan a unos dos milímetros del central y atraviezan los mismos tejidos. Al salir por debajo de la inserción tendinosa del trozo de músculo que hemos cortado se desvían hacia el ecuador del globo ocular, para atravesar en este punto algunas fibras escleróticas. Después de haberlas atravesado convergen en dirección al central a pasando por la parte interna del músculo, cápsula y conjuntiva, viene a terminar en la misma región del punto central. Estos puntos de sutura pueden oblicuarse mas o menos, hacia arriba o hacia abajo, si el estrabismo es algo superior o inferior.

Constituida la sutura en esta forma limita una superficie romboidal con un diámetro anteroposterior mayor que el vertical. Actúan en ella tres fuerzas sobre la línea que une los tres puntos de intersección del músculo con la esclerótica, produciéndose así una composición de fuerzas cuya resultante dependerá del valor de cada una de ellas, o sea de la potencia de cada una de estas cuerdas. Su colocación especial da lugar a fuertes adherencias escleróticas, que impiden con el tiempo vuelva a presentarse de nuevo la desviación ocular, como ocurre con las suturas que se practican en otros procedimientos, proque no debemos olvidar que la colocación de los puntos es uno de los factores principales de la operación.

Colocados en la forma indicada los puntos de sutura, se suelta la pinza de Prince, y teniendo los hilos de seda algo tensos un ayudante, se secciona la porción reseca del músculo al ras de su inserción tendinosa. Después, aproximando el ayudante con las pinzas los bordes de la incisión de la conjuntiva, se hace el anudamiento de los puntos, comenzando por el del centro. Al colocar éstos, se trae el borde cruento del músculo reseca a injertarle en el punto de la esclerótica, donde se insertaba el tendón, provocando, por lo tanto, un *avanzamiento indirecto del músculo*, puesto que una parte más posterior del mismo viene a colocarse en una región más anterior de la esclerótica, pero sin traspasar los límites de la primitiva inserción tendinosa, con el fin no se rompa el equilibrio muscular. Si el estrabismo es directo, se aprietan igual todos los puntos, pero si es ligeramente oblicuo, pondremos un poco más tenso el lateral correspondiente.

Hecho esto, se fijan los extremos de la seda con un esparadrapo a la

parte externa, se instilan en ambos ojos unas gotas de atropina con el fin de paralizar la acomodación, y se coloca un vendaje binocular. Los grabados que publicamos hacen que se comprendan fácilmente los distintos tiempos de la nueva técnica operatoria que empleamos para corregir el estrabismo.

Si hemos seguido todas las reglas de la asepsia, no hay temo a la infección, y, por lo tanto, no es necesario levantar el vendaje hasta pasadas las cuarenta y ocho horas después de la operación. Al levantar el vendaje lavaremos bien ambos ojos con la solución bórica e instilaremos nuevamente atropina. Estas curas se repetirán cada dos días, y al octavo se podrán quitar los puntos de sutura, dejando un vendaje monocular en el ojo que no se desviaba, y en el estrábico una pantalla colocada más hacia el lado que tuvo el estrabismo, para que el enfermo mire por el lado contrario. Se sigue instilando atropina hasta que desaparezca por completo todo fenómeno de reacción operatoria, y llegado este momento se suprime el midriásico para que vuelvan a contraerse las pupilas, conseguido lo cual se hace la dirección del defecto de refracción que hubiere en los ojos y se da comienzo a la gimnasia de convergencia y acomodación, que ejecutará el enfermo por espacio de mucho tiempo, empleando los medios ortópticos, con el fin de conseguir la visión binocular, que es la verdadera curación del estrabismo.

Desde el año 1915, que empecé a practicar esta técnica operatoria, hasta la época presente, la he modificado en algunos detalles para corregir pequeñas deficiencias que pude observar tenía en mis primeras intervenciones. En la descripción que dejo expuesta, se incluyen hasta los datos más insignificantes de las modificaciones hechas en el procedimiento operatorio hasta el momento actual. Creo se puede decir que he llegado a su perfeccionamiento completo, si tenemos en cuenta los resultados altamente satisfactorios que obtengo con su empleo.

El número de enfermos que he sometido a esta intervención quirúrgica desde el año 1915 al 1919 pasan de 100.

¿Cómo podría evitarse la intervención quirúrgica en el estrabismo? En muchos casos previniendo el desarrollo de la enfermedad, para lo que es necesario tratarlo convenientemente cuando se inicia, porque hemos de advertir que el tratamiento quirúrgico no produce siempre la curación verdadera del estrabismo. Es, por lo tanto, de un valor terapéutico inferior a los medios ópticos y ortópticos.

Los primeros corrigen los vicios de refracción del aparato dióptrico,

o lo que es lo mismo, evitan las causas que predisponen al estrabismo, y los segundos educando la visión binocular contribuyen poderosamente a remediar el defecto de la facultad de fusión, de donde resulta, en realidad, que el enderezamiento ocular mediante la intervención quirúrgica, no es más que un auxiliar de los medios funcionales cuando buscamos la curación verdadera del estrabismo.

Si el estrabismo es un vicio de desarrollo de la visión binocular, que se favorece por causas locales, se comprende fácilmente que el empleo de los llamados medios funcionales debe establecerse desde el momento que se inicia el estrabismo en la niñez. Es preciso educar a los padres para que salgan de su indiferencia injustificable, esperando una curación espontánea que nunca llega, y es necesario convencer a las madres de que es imprescindible hacer cuanto sea posible para que los niños lleven cristales correctores de su vicio de refracción, aún cuando sean antiestéticos.

Si los padres abandonan a sus hijos en la niñez, las modificaciones que se realizan en el aparato de la visión binocular son cada vez más profundas, y cuando son ya crecidos, la corrección óptica y el tratamiento ortóptico darán un resultado poco satisfactorio, teniendo necesidad de asociarlo al tratamiento quirúrgico.

La intervención operatoria no está justificada en los niños pequeños de dos a siete años. En esta edad limitaremos nuestro tratamiento a una corrección óptica bien hecha, a la atropinación del empleo de los ejercicios ortópticos desde el momento que lo permita la inteligencia del niño, con lo que se consigue muchas veces desarrollar la facultad de la fusión, manteniendo el equilibrio monocular. Estos ejercicios los hará el niño lo antes posible con el amblioscopia Worth, instrumento el más apropiado para el niño porque le divierte, al mismo tiempo que le hace desarrollar el sentido de la fusión.

Cuando los padres han abandonado al enfermito, en los primeros tiempos de presentarse la enfermedad, y el estrabismo se ha hecho permanente con marcada ambliopia del ojo desviado, es preciso mejorar la agudeza visual de éste, corrigiendo la anatrópia atropinizando el ojo fijador. Desde el momento que el ojo ambliope trabaja diariamente, mejora su agudeza de una manera rápida, y en poco tiempo nos encontramos en condiciones de practicar con fruto ejercicios de visión simultánea y binocular, con el amblioscopia y el esteroscopia.

El mismo adolescente, que sabe leer, debe someterse a los medios funcionales que dejo indicados y además hará diariamente sesiones

de visión binocular ayudado del diplóscopo de Remy, alternando los ejercicios eteróscopicos con cartones divertidos. El uso del esteróscopo, dice Onfray, es muy útil, después de hacer uso del diplóscopo "para relajar activamente la convergencia, fortificar el sentido de la fusión y producir la sensación de relieve"; los ejercicios ortópticos deberán terminarse con sesiones de visión binocular sin instrumento, mediante la lectura comprobada. El estrábico debe trabajar constantemente por extender su campo de visión binocular, y fortificar sin descanso la aptitud adquirida de fusionar la doble imagen, para lo que es preciso que ejerzamos sobre él una especie de sugestión y no abandone el tratamiento.

Aún cuando no llegamos al enderezamiento del globo ocular, la aptitud a la fusión de las imágenes que hayamos conseguido por medio de los ejercicios funcionales, contribuirá poderosamente a la curación verdadera del estrabismo cuando tengamos necesidad de intervenir quirúrgicamente.

Si a pesar de haber empleado todos los medios llamados funcionales, el niño llega a los doce o catorce años sin haber conseguido la visión más que en ciertas posiciones de la mirada y sin haber corregido más que de una manera incompleta su desviación, debemos aconsejarle la intervención quirúrgica, que aplicaremos sobre los dos ojos, con el fin de obtener su curación verdadera.

La técnica quirúrgica que apliquemos ha de variar necesariamente según el grado de desviación estrábica que tenga el enfermo. Si ésta es débil, inferior a 14° , es suficiente el doble avance muscular indirecto con pequeña resección tendinosa y la sutura en rombo, para que podamos conseguir fácilmente la corrección del estrabismo. Cuando el ángulo de desviación alcanza de 18 a 20° , será preciso además del avance, la resección muscular más extensa en la forma indicada anteriormente, y por último, para las desviaciones más elevadas hasta cuarenta y tantos grados, asociaremos al avance y resección, la debilitación de los músculos antagonistas, rectos, internos o externos, según sea el estrabismo convergente o divergente, practicando en todos los casos la sutura romboidal.

No olvidaremos en la técnica operatoria un detalle que, a mi juicio, tiene mucha importancia; me refiero a que no debemos quietar los hilos de sutura mientras no pasen ocho días después de la intervención para que las adherencias de los tejidos puestos en contacto durante el acto operatorio se consoliden completamente antes de separar los hilos. Es necesario también recordar el beneficio que reporta la

atropinización de los dos ojos para relajar la convergencia, el empleo inmediato de los ejercicios ortópticos, mediante el diplóscopto, el esteróscopto y los ejercicios de visión binocular sin instrumentos, en el último período. Mediante estos recursos, podemos en muchos casos, si el enfermo dispone de tiempo y paciencia, llegar a la verdadera curación del estrabismo.

Decimos al hablar de la intervención quirúrgica, que debe aplicarse sobre los dos ojos, y así debe ser en efecto, cuando buscamos la curación verdadera; pero en aquellos casos en los que el enfermo no desee recobrar su visión binocular, o se trate de una muchacha joven, que bajo ningún concepto ha de llevar puestos sus vidrios correctores, para no afeár su belleza, estamos autorizados a practicar una intervención monocular y hasta la tenotomía *prudente*, operación que hemos de rechazar en la mayoría de los casos, por las razones que hemos consignado al hablar de la técnica quirúrgica del estrabismo.

De cuanto llevo expuesto se deducen los siguientes corolarios:

El estudio de la técnica operatoria del estrabismo, es uno de los capítulos de nuestra especialidad que menos ha progresado en España, hasta la época actual, lo que se debe a la escasez de material clínico humano para intervenir quirúrgicamente, y no al prejuicio religioso, como opina algún ilustre colega extranjero.

Las intervenciones quirúrgicas del estrabismo son diversas, según dejamos consignado. La mayor parte de los autores emplean una técnica que se diferencia de la general, en detalles más o menos importantes, sin que tengamos hasta la fecha una que sea aceptada como procedimiento ideal por todos los operadores.

La tenotomía, el acortamiento muscular, el avanzamiento muscular, el avance capsular, cápsulomuscular, el muscular simple, o con resección de mayor o menor parte del músculo, y la miectomía, son los distintos procedimientos que aisladamente, o combinados con la tenotomía del músculo antagonista, se vienen aconsejando indistintamente según la variedad y mayor o menor grado de desviación que tenga el enfermo estrábico, lo que demuestra la falta de un procedimiento quirúrgico que pueda aplicarse con las variantes consiguientes, en todos los casos de estrabismo que se nos presenten.

La tenotomía, operación indosificable, de efectos variables, inseguros y perniciosos siempre para la convergencia, debe abandonarse casi por completo, practicándose solamente en algún caso muy excepcional, porque puede producir a la larga fecha la retracción del

pliegue semilunar de la carúncula, desviaciones secundarias, paresia muscular y dificultades en la visión binocular.

El avanzamiento muscular, o el acortamiento del músculo, que aconsejan practicar algunos autores unilateralmente, es insuficiente en la mayoría de los estrábicos, si no se combina con la tenotomía, en cuyo caso la intervención adolece de los defectos de esta operación. Lo mismo ocurre con los desbridamientos capsulares, el avanzamiento muscular sencillo monocular asociado con la resección del músculo.

Son procedimientos operatorios insuficientes por sí solos para corregir los diversos grados de una desviación estrábica pronunciada.

El doble avanzamiento muscular sencillo *indirecto* o con resección de cierta cantidad de músculo, siempre asociado a la *sutura romboidal* y combinado con la *debilitación de los músculos antagonistas* cuando la desviación estrábica es pronunciada, me parece el tratamiento quirúrgico ideal del estrabismo en la actualidad.

Las desviaciones estrábicas de poca intensidad se corrigen con el doble avanzamiento sencillo indirecto, pequeña resección tendinosa y la sutura en forma romboidal. En los grados fuertes de estrabismo es preciso añadir además, la resección muscular en mayor o menor extensión, y la debilitación de los músculos antagonistas.

Con este tratamiento quirúrgico, la corrección estética, conseguida desde el primer momento, persiste y mejora en el porvenir al restablecerse el equilibrio muscular, lo que no ocurre generalmente con otros procedimientos.

Es la operación mas racional para que se normalicen paulatinamente las excursiones del globo ocular y para que la visión binocular se restablezca en todos aquellos casos que lo permita su agudeza visual.

Los operados deben someterse a la corrección óptica y al tratamiento ortóptico consecutivo, como en los demás procedimientos operatorios.

El doble avance muscular *indirecto*, la *sutura romboidal* y la *debilitación* del músculo antagonista, son los tres factores importantísimos que nos autorizan a considerar como nuevo el procedimiento quirúrgico que practicamos nosotros, para corregir el estrabismo.

A NEW PROCEDURE IN THE EXCISION METHOD OF PTERYGIUM OPERATION

DR. E. CAMPODONICO

Lima, Perú

It is a well-known fact that pterygium is of frequent occurrence in the tropics as compared with its relative rarity in the temperate regions. In the coastal zone of Peru it is especially prevalent, owing to the dusty condition of the air consequent on the dryness of the soil and the lack of rainfall.

In my one-year-long visit to the clinics of the United States I have seen very few operations for pterygium, whereas here in Peru it is an operation of almost daily occurrence. My experience in the European clinics points to the same infrequency.

With such an ample field and variety of cases as are forthcoming in the Peruvian ophthalmic clinics I had the opportunity of lavishly trying all the known methods, gauging their advantages and disadvantages as well. Indeed, our outdoor patients' consulting department of the Italian hospital in Lima has an average daily number of 150 ophthalmic cases. In the following exposition I beg to outline a procedure which has given me the best results for a period of over twenty years.

The needed instruments are: lid-speculum, small fixation forceps, —preferably one-toothed,—angular keratome, straight or curved fine scissors, fine silk suture, and occasionally a needle-holder.

The operation is performed in three steps: (1) Dissection of the pterygium's head out of the cornea and adjacent sclera by means of the angular keratome; (2) excision of the pterygium's head and body by means of small scissors; (3) suturing of the flaps.

I have nothing to say about the first and second steps, as the performance thereof is a matter of routine practice. The only thing I should insist upon is the use of the anesthetic by *instillation*, not by subconjunctival injection, as the areolar infiltration resulting from the injection is detrimental to the accurate subsequent coaptation of the flaps.

The third step, *i. e.*, suturing, is the one wherewith I shall deal with some detail on account of the decisive influence on the outcome and ultimate issue of the whole operation.

The placing of the conjunctival suture and cicatrix in a position favorable for recurrence is the paramount disadvantage of most of the methods in vogue for the extirpation of pterygium. The safest plan is to remove such conjunctival union from exposure in the



Fig. 1.—Conjunctival defect after excision.

palpebral fissure, as by so doing, in the excision operation, we actually lessen the proclivity to recurrence.

Fig. 1 shows the conjunctival defect after excision of the pterygium.

Our method consists in suturing the conjunctiva to the point *a* with the episcleral tissue and conjunctiva of the point *b*, as is shown in Fig. 2. Next the points *c* and *d* of conjunctiva are united by another suture, as shown in Fig. 3.

As is easy to understand, the essential feature of this method is the insertion of a suture which is passed through the conjunctiva of one flap at a point *a* (Fig. 1), and through the episcleral tissue and con-

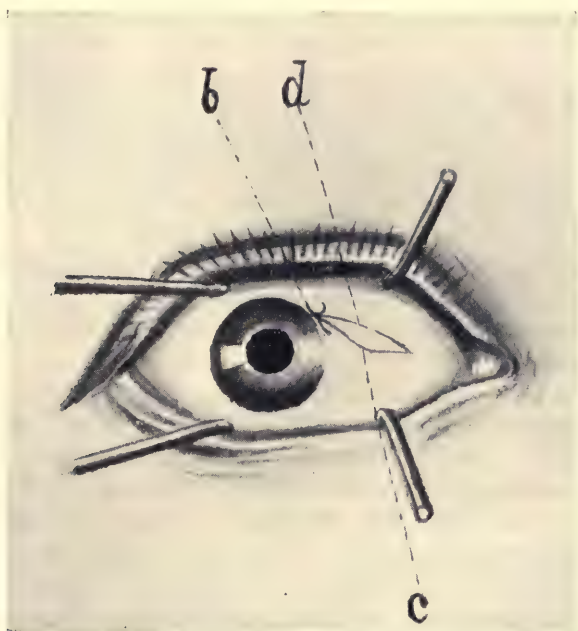


Fig. 2.—Conjunctiva sutured in place.

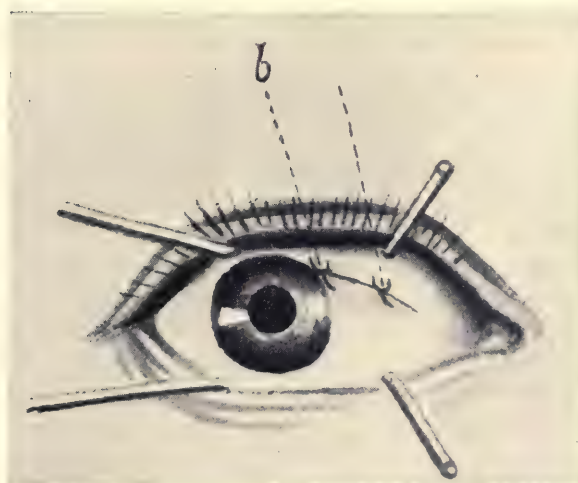


Fig. 3.—Second conjunctival suture.

junctiva of the point *b*. The points *a* and *b* (Fig. 1) are equidistant from *y*. Thus, when *a* comes to *b*, the tract *a y* overlaps the portion of limbus *b y*. The point *a* is perfectly movable, because it takes the conjunctiva only, whereas the point *b* is quite steady and immovable by reason of comprising the limbus conjunctivæ and episclera as well.

Occasionally, and especially in cases when the pterygium's head is large, it is advisable to undermine a little the flap *a* in order to avoid the stretch on this flap when *a* is anchored to *b*.

On drawing the flap *a* to *b* an actual overlapping of a portion of the cornea may result; this, however, is quite uneventful, and on taking off the bandage in a couple of days' time everything will be found in a perfect coaptation.

Instead of making the lower flap movable and the upper one fixed, we may just as well do the reverse and anchor a loose point of the upper flap to the episcleral tissue and limbus conjunctivæ of the lower. This we have done several times, namely, in cases in which it is easier to loosen and slide the upper flap than the lower. We prefer, however, the first way, because the conjunctival seam is thus more efficiently protected by the agency of the upper lid from exposure. At the end of the operation it will be found also that fibers which had before a horizontal direction, now have a vertical one; their natural trend of growing is consequently thwarted.

A still further advantage in removing the conjunctival apposition line from the horizontal meridian is the fact that by so doing we eliminate in the horizontal line the presence of sutures which undoubtedly act as a stimulus for reproduction of connective and fibrous cicatricial tissue, just exactly in a place where it is least desirable.

The eye is bandaged for seven or eight days, after which the sutures are removed. The bandage is changed every other day. Whenever possible, it is a good plan to keep both eyes bandaged at least for a couple of days.

As previously stated, in a place like Lima, where pterygia are especially plentiful, I have operated many hundreds of cases by the procedure just described and have eagerly and abundantly tried every new method that reached my acquaintance, with the result that I always gave it up to return to the simple course I have outlined above.

SUMMARY

The most salient disadvantage in the ordinary excision method of pterygium operation is the placing of the conjunctival suture and

cicatrix in the horizontal meridian, *i. e.*, in a position especially favorable for recurrence.

The author's procedure consists in removing the suture and cicatrix as much as possible from the palpebral fissure by simply anchoring a movable and nearly middle point of the lower conjunctival flap to a fixed and steady point of the upper flap, namely, *to the episcleral tissue and limbus conjunctivæ of the upper flap*. Thus the limbus corneæ, from which the pterygium's fibers have been extirpated, is covered by a sound stretch of conjunctiva and the horizontal trend of the fibers is thwarted vertically.

THE SUBCONJUNCTIVAL EXCISION OF PTERYGIUM

DR. S. LEWIS ZIEGLER

Philadelphia

That pterygium is a vascularized, fleshy growth of conjunctiva invading the cornea, and composed chiefly of *hypertrophied subconjunctival tissue*, is a pathologic fact which is highly significant in its relation to both treatment and prognosis. The ancients regarded pterygium as malignant in origin, and, like other neoplasms, they removed it by total ablation. Scarpa¹ was the first to deny this origin, but classified it as a varicosity resulting from chronic conjunctivitis.

Alt² hypothetically ascribed the origin of pterygium to a marginal corneal ulcer, although he could not demonstrate this in his preparations. Goldzieher³ found considerable disturbance of the corneal epithelium around the head, while beneath it Bowman's membrane was detached and the fibers of the corneal stroma were thickened. At the margins he described small cavities dotted with epithelium and other signs of mucous degeneration. He also noted episcleral thickening, and believed that corneal ulceration was the origin.

Horner⁴ first promulgated the view that pinguecula and pterygium were two stages of a similar pathologic process. Poncet,⁵ in a recent specimen, found no evidence of corneal ulceration, although Bowman's membrane was slightly eroded and the vessels somewhat distorted; but there was no sign of inflammatory proliferation. Although neoformation of tissue was absent, he did observe that the elements of a cicatrix were present. In a few preparations he found

vibrios and spores which he thought might be responsible for the focal point of the affection in these individual cases.

Fuchs,⁶ in an extensive study of several hundred specimens, both *in vivo* and postmortem, eliminated ulceration and supported the view that pinguecula was the origin. He had never found the vibrios noted by Poncet, nor were other microorganisms observed. He occasionally found yellowish spots with hyaline aggregation, and in six cases he noted small cystic cavities filled with a clear liquid. De Schweinitz,⁷ Strachow,⁸ and others have confirmed this observation by reporting similar cases of cystic formation within the body of the pterygium.

Panas,⁹ in reviewing the pathology of pterygium, called attention to its macroscopic resemblance to cicatricial tissue. He further declared that the theory of corneal ulceration is founded neither on clinical observation nor on histologic examination. He concluded that the primary lesion was an involutive dystrophy of the cornea with secondary hyperemia, hypertrophy, and retraction of the conjunctiva; that there was a predisposing cause in the individual, to which were added exciting causes due to prolonged exposure to irritants, such as dust, sand, heat, wind, or lacrimal disturbances.

Pterygium should always be removed—(a) When it is progressive; (b) when it interferes with vision; (c) when it limits ocular motility; (d) when a capital operation on the globe is planned; or (e) when it is cosmetically disfiguring.

The requisites necessary to success in the operative treatment of pterygium are: (1) Thorough removal of the head, leaving clear corneal tissue; (2) careful excision of the subconjunctival tissue, especially about the limbus; (3) complete closure of the conjunctival wound, with close approximation to the limbus; (4) the avoidance of conjunctival tearing and tension; (5) rapid primary union, with as thin a conjunctival flap as it is possible to secure.

The earliest methods resorted to for the removal of pterygium depended upon *excision*, more or less complete. The resulting wound was universally left open, to be healed by granulation. This neglect was responsible for many cases of recurrence. The necessity for a complete covering of the scleral defect cannot be too strongly emphasized as a precautionary measure to obviate such a danger. It is better to insert a suture close up to the limbus, and if necessary, to include the scleral and episcleral tissue, in order to fix it there evenly and thus promote firm, smooth healing. Neglect of this simple

technic will often permit the formation of cicatricial tissue, which will not only interfere with the motility of the subjacent rectus muscle, but may encourage recurrent or pterygoid growth.

Another defect in many of the older operations arose from the neglect to remove the head of the growth, which, if progressive, continued to encroach upon the cornea. Many methods have been devised for the successful accomplishment of this simple essential, and a variety of instruments have been suggested. A narrow von Graefe cataract knife is the knife of choice adopted by the majority of operators. As a rule, this is entered flatwise beneath the constricted neck, with the edge turned forward and the head cleanly shaved off from the cornea. Others select Beer's knife for the same purpose, because its angular point makes it easier to insert, and because the wider blade yields a clean, broad incision that is safer to make and freer from post-operative tags of tissue. Personally, I prefer to use Beer's knife as a dull dissector, first making firm traction on the neck with forceps and then applying the back of the blade near its point to the stretched-out marginal fibers. These will gradually yield, just as a lichen is pulled off from a tree, and a clean corneal surface is secured. If, by chance, a small shred of tissue is left behind, the knife should be reversed and the blade applied as a razor to cleanly shave the corneal surface. The ordinary broad-pointed scalpel is often used in a similar manner. Some choose the blunt-pointed knife of Desmarres. Others prefer to use the angular keratome or Taylor's broad needle to accomplish a like purpose, the value of the latter having been strongly urged by Black.¹⁰ A bent, double-edged, short von Graefe knife has been adopted by McReynolds,¹¹ but this again is practically identical with the angular broad needle of Taylor. McReynolds insists that *shaving* the cornea is far more effective in his climate than *divulsion*.

Removal of the head by *blunt dissection* has been warmly advocated by many. Prince¹² has particularly recommended *divulsion* with a strabismus hook applied successively to small sections on each side of the growth, which is torn loose by short, jerky movements from the neck toward the apex, until all the filaments are severed and the cornea stripped clear. The strabismus hook "divulsor" has also been made up with a beveled edge or with a knife edge, and also with a sharp point. Special hooked scissors, such as Chadwick's, have been devised for this purpose, but many simply use the closed blades of the tenotomy scissors as a blunt dissector. The multi-

plicity of instruments shows that there is no really important advantage to be gained from the use of one over the other; all of which emphasizes the fact that the choice of an instrument is a mere matter of detail in the technic of each individual operator.

Curetment of the corneal surface should be gently made if any apical fibers remain adherent. The Gallardo corneal curet, the small toothed curets of Skeel or Ziegler, and the hoe-shaped corneal spud have all been employed for this purpose. Both the knives of Beer and of von Graefe have been used to shave the surface of the cornea.

The galvanocautery, as suggested by Panas, may be lightly applied to the rough corneal surface if other measures should fail to clear it. This is of particular advantage where the growth is so adherent as to require morcellation. The eschar must be promptly removed with the curet and $\frac{1}{2}$ per cent. formalin carefully applied to prevent leukomatous scarring.

Accepting the theory that *subconjunctival hypertrophy* is more or less *cicatricial* in character and possesses, therefore, a persistent tendency to undergo contraction, and that it is this tissue which invades the cornea and causes recurrence, I conceived the idea that the thorough removal of this subconjunctival tissue would not only encourage smooth healing, but would also eliminate the danger of recurrence. It is likewise my belief that the same principle holds good in the surgery of symblepharon or other growth of a cicatricial character. Acting on this theory, I began, in 1890, to practise the following simplified method of subconjunctival excision, which in my hands has proved to be not only a cosmetic success, but has been absolutely free from recurrence.

Cocain is sufficient as an anesthetic, and adrenalin may be instilled as a hemostatic. If desired, a stronger solution of cocain (20 per cent.) can be applied directly to the growth with an applicator while the lids are held open to avoid contact with the cornea. Some prefer to inject novocain, 1 per cent., beneath the field of operation, but the consequent edema may distort the outlines of the growth.

AUTHOR'S METHOD OF SUBCONJUNCTIVAL EXCISION

First Stage.—The neck of the pterygium is grasped firmly with rat-tooth fixation forceps, drawn tense, and the marginal fibers of the head freely divided with the back and point of a Beer's knife, used as a dull dissector. If the apex is unusually adherent, the knife may be passed beneath the neck and the head shaved off (Fig. 1).

Second Stage.—Still grasping the apex and holding it tense, both sides of the pterygium are cut loose with the conjunctival scissors, the body undermined, and the flap lifted up (Fig. 2).

Third Stage.—The forceps which grasp the apex are now handed to

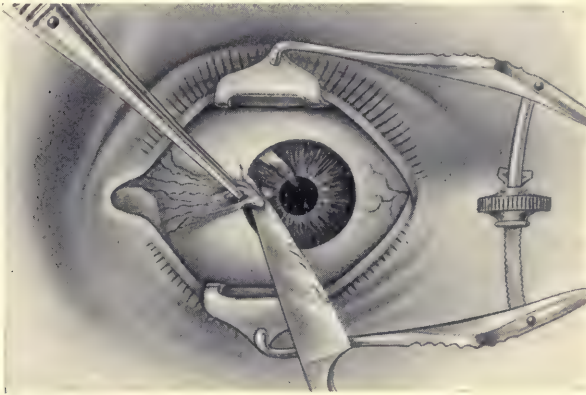


Fig. 1.—First Stage: Head removed with Beer's knife as dull dissector.

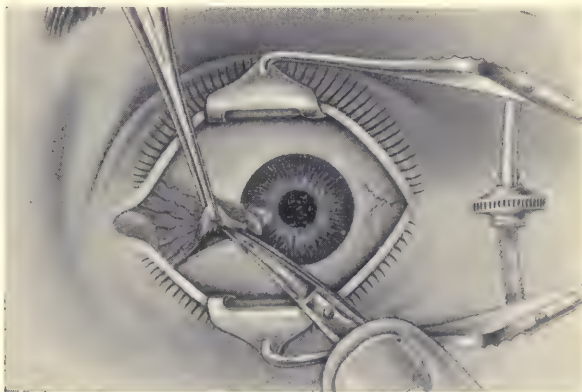


Fig. 2.—Second Stage: Detaching body from sclera with scissors.

an assistant, who continues to hold the flap taut, while the *subconjunctival tissue beneath the flap* is grasped by a second pair of forceps and carefully dissected off from the conjunctiva by delicate snips of the sharp-pointed conjunctival scissors (Fig. 3). When the conjunctival flap is freed from the underlying tissue its *apex is excised* and

the remaining flap of pure conjunctiva dropped back toward the canthus.

Fourth Stage.—According to the size of the scleral defect to be covered I resort to one of three different procedures—(a), (b), or (c):

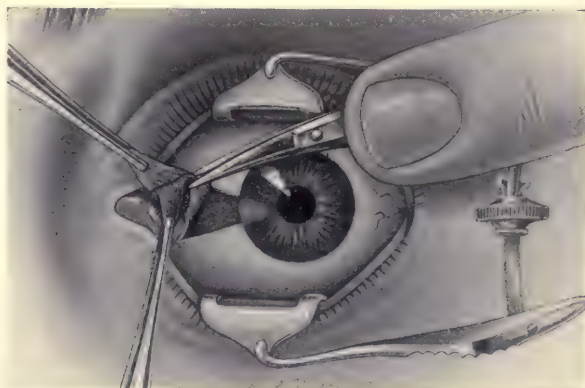


Fig. 3.—Third Stage: Hypertrophied subconjunctival tissue being carefully dissected from conjunctiva with scissors.

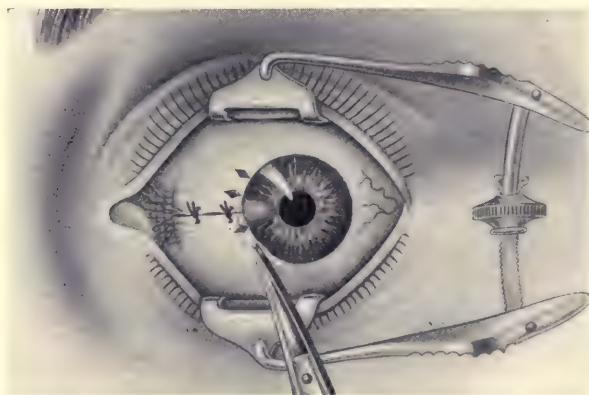


Fig. 4.—Fourth Stage: Sutures inserted. Juxtacorneal one anchored in sclera. Roll of conjunctiva snipped to relieve tension and smooth it out.

(a) If the pterygium is small, the conjunctival wound is closed by two sutures, the juxtacorneal one being anchored in the sclera. This may cause overlapping of the corneal margin, which is corrected by clipping the cuff-like roll of conjunctiva in several places with scissors

and pushing it back. As the conjunctiva again smooths out these "pie-cuts" open into small, diamond-shaped perforations which relieve all tension and heal smoothly (Fig. 4).

(b) *If the wound is larger and the conjunctival tension is great*, two short paracorneal incisions (5 mm.) are made, above and below, the conjunctival edges undermined, and the wound united by two sutures, the juxtacorneal one being anchored in the sclera.

(c) *If the denuded area is extremely large*, two sets of liberating incisions are made, the paracorneal incisions above and below being supplemented by incisions made parallel to the first (5 to 10 mm.), and placed at the canthal extremity of the wound. The quadrilateral flaps thus formed are closed with three sutures, the juxtacorneal one being anchored in the sclera, and a mattress anchor suture placed at the canthal intersection, as in Knapp's double transplantation.

AFTER-TREATMENT.—As a rule, the simple technic of the first procedure (a) is wholly adequate to close the wound. Healing is smooth and prompt, owing to the avoidance of extensive dissection and the freedom from tension. A monocular dressing is applied for three or four days. The Liebreich patch may be worn a day or two longer. The stitches can be removed on the third or fourth day. Boric acid irrigation is used once daily until the pad is discarded, then three times a day. The eye usually remains red for one or two weeks. If there is lacrimal disturbance, the tear-duct should be dilated at least ten days before the pterygium operation is attempted.

The principle of subconjunctival excision of the hypertrophied adventitious tissue can be applied as well to any other of the well-known pterygium procedures. I have tried it in McReynolds' modification of the technic of Desmarres, Sr., and found it successful in producing a much thinner flap, which made the procedure a greater success.

To recapitulate briefly the advantages of subconjunctival excision of pterygium, I should say that the four factors most worthy of consideration are—(1) Reduction of a thickened flap; (2) avoidance of extensive dissection; (3) freedom from conjunctival tension, and (4) prevention of recurrence.

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DISCUSSION OF PAPERS OF DRS. CAMPODONICO AND ZIEGLER

DR. JOHN O. McREYNOLDS (Dallas, Texas): In discussing the papers of Drs. Ziegler and Campodonico three distinct observations are relevant:

1. For small and medium size pterygia in localities that do not strongly predispose to the development or recurrence of the growth many different surgical measures may yield excellent results. The crucial test is made, however, in the large vascular types associated with atmospheric conditions favorable for the production of this condition.

2. The second observation relates to the removal of the head of the pterygium from the cornea. In my experience the essential thing is an absolutely complete and smooth removal of every vestige of the head, leaving only smooth clear corneal tissue beneath. This eliminates all forms of divulsion, curetting and cauterization, and requires a dependable cutting instrument. For ordinary cases a Graefe knife is sufficient, but for exceedingly large pterygia, covering practically $\frac{9}{10}$ of the cornea, the most effective instrument is a very sharp angular keratome not broader than 2 mm. at its base. This permits the surgeon to apply the cutting edge in such a way to the spherical surface of the cornea that it can shave off every particle of the growth without going too deep into the corneal stroma.

3. The third observation relates to the removal and disposition of the body of the growth with the necessary covering of the denuded area of the sclera.

Each of the papers emphasizes a feature of value. The paper of Dr. Ziegler dwells upon the importance of removing the redundant subconjunctival tissue. The paper of Dr. Campodonico dwells upon the importance of eliminating all sutures in the palpebral opening. Both of these objects are worthy of every consideration and both can be accomplished by means of a single suture which fixes the growth in the lower cul-de-sac, utilizing the attenuated conjunctival element of the pterygium in covering the scleral defect, taking care that there shall be no break along the superior border of the growth which would allow the wound to gape and necessitate the introduction of sutures in the palpebral opening.

DR. A. E. PRINCE (Springfield, Ill.): The subconjunctival removal of pterygium is a practice which I have followed for many years in cases where there is a large amount of hypertrophied tissue, and this I have done by simply excising the fleshy portion after laying back the superficial conjunctiva.

I wish particularly to speak of a method of treating pterygium in another class of cases; those which are attenuated, spread over considerable area, or

are very minute, and have no flesh under them. They hardly justify a surgical operation if it can be avoided. In those cases I have found that by taking some carbon dioxide ice, having previously cocaineized the cornea, and applying the ice at intervals of about a week, the pterygium will disappear. I think what takes place is a destruction of the blood globules in the minute capillaries and subsequent atrophy of the tissue. In an old lady, where three-fourths of the cornea was covered and vision reduced to 1/200, four treatments brought vision to 20/200. The application should be one second.

Carbon dioxide is simple to handle. You can get a tank of the gas in almost every city where there is a soda fountain. Suspend it on the wall with the portion at which the faucets are attached at an angle of about twenty degrees, so that all of the impurities will be at the top and the liquid come to the bottom. Have a plumber make a little reducer, the fine end of which will fit an ear speculum. The ear speculum is then put into this reducer, and a napkin, folded about eight thicknesses, is held securely over the ear speculum. Upon opening the faucet at the end of the tank the liquid carbon dioxide fills the ear speculum and the portion which escapes in gaseous form through the folded napkin freezes that which remains in the ear speculum and is a solid cast of carbon dioxide ice. This can be made to take any shape desired by cutting it with a knife or scissors. This use of carbon dioxide ice for pterygia was described in *Archives of Ophthalmology* in the year 1916, under the caption, "Carbon Dioxide Ice in the Treatment of Trachoma, Vernal Catarrh, and Pterygiums." I wish to add a previous report of a case which proved intractable after six attempts at excision, and was entirely cured by eight weekly applications of the carbon dioxide ice.

DR. S. LEWIS ZIEGLER (closing): I have not had an opportunity of trying the carbon dioxide snow, recommended by Dr. Prince, but it is a good suggestion for the attenuated cases; in the fleshy cases I have sometimes resorted to galvano-cautery puncture similar to the operation I once suggested for ectropion, except, of course, that you pass your needle, which must be a fine one, parallel with the surface of the globe instead of passing the tip into the tissue vertically, because you must not cauterize the sclera, since Knapp has shown there is danger of disturbance of the aqueous and ultimate cataract. You can also resort to electrolysis. Another method is high frequency desiccation, as practised by Dr. Clark, of Philadelphia. In some cases of keloid that has been of value.

The manner of removing the head of the pterygium is an individual question. I happen to prefer Beer's knife; Dr. McReynolds uses the broad needle, and Dr. Prince practises divulsion—one selects that which is most effective in his own hands. Where there is morcellation following removal of the growth it may be necessary to cauterize the small particles left. If you do and wish to avoid leucoma of the cornea, it would be wise to remove the eschar by curetting and applying weak formalin, one-half of one per cent.

Slicing off the cornea has been advocated by some and criticized by others. Dr. McReynolds thinks in his climate it is most successful. In the literature are cases in which severe ulceration has followed, so that again is a matter of individual choice. The French school generally favors the galvano-cautery.

They cauterize only the area of the cornea where the head has been removed.

In regard to cases where there is considerable ulceration of the cornea after operation, I think the suggestion of Dr. Pontius, of Philadelphia,¹ in regard to the use of full strength adrenalin for regeneration of the cornea, is an excellent one. It has proved most successful in my hands.

LA TUBERCULOSIS OCULAR INFANTIL

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En la patología ocular del niño, el capítulo de la tuberculosis aparece estudiado de un modo incompleto, sobre todo en lo que se refiere a la comprobación anatómica-patológica de las lesiones.

De nuestro material clínico hemos utilizado tres casos, en los que fué necesario practicar la enucleación por lesiones graves y como medio profiláctico y terapéutico.

El estudio histológico de estos globos oculares es el material utilizado para esta conferencia, pues en ellos hemos podido determinar procesos tuberculosos como etiológicos de los graves trastornos que presentaban a nuestra observación clínica.

Los procesos tuberculosos primarios del globo ocular ofrece para el diagnóstico clínico gran dificultad, pueden confundirse con lesiones inflamatorias de tipo crónico, y no presentan estigmas precisos para su diagnóstico diferencial, esto unido a que pueden no existir manifestaciones en ninguna otra parte del organismo, ausencia de antecedentes familiares reacción de Pirquet negativa, ningún antecedente morboso ocular.

Las manifestaciones secundarias corresponden con frecuencia a enfermos de tuberculosis intestinal y pulmonar, existe antecedentes familiares, reacción de pirquet positiva, terreno abonado a la infección, ataques repetidos de conjuntivitis pustulosa o flictenular, en esta forma el diagnóstico clínico es más sencillo.

No haremos ahora la descripción de síntomas clínicos tan variados como ofrece la tuberculosis ocular. En el ojo humano constituido por tres capas de naturaleza tan distinta como—córnea, esclerótica, iris, ciliar coroides, retina y óptico—es posible observar formas simples de infección de estas membranas o combinaciones.

Siempre que es un proceso crónico o agudo, no obtengamos mejoría por los medios terapéuticos corrientes y la investigación de otro tipo

¹ N. Y. Med. Jour., 1912, p. 637.

de infección resulte negativa debemos pensar en una lesión tuberculosa.

Cuando el globo ocular es atacado de tuberculosis se destruye antes de generalizarse la infección, en un mismo globo es posible encontrar desde la zona inflamatoria o productiva, hasta la zona gaseosa; zonas de propagación, lesiones más avanzadas o intensas en relación a la resistencia de la membrana.

No podemos excluir la posibilidad de la generalización si tenemos en cuenta la emigración bacilar por vías sanguíneas, solo favorece a la localización ocular la capilaridad de los vasos en los plexos arteriales y venosos, los bacilos que resisten al ataque de los leucocitos polinucleares, quedan detenidos en los vasos por un trombus fibrinosos.

Nuestro estudio va a quedar limitado al globo ocular propiamente dicho prescindiendo de los anejos, y se las manifestaciones tuberculosa de la conjuntiva.

El problema terapéutico de la tuberculosis ocular queda reducido a las manifestaciones primarias, debe intentarse un tratamiento general apropiado a estos casos, el local en relación a la sintomatología, si nada se consigue, así el proceso avanza y la función visual está completamente abolida, se impone la enucleación como medio profiláctico.

HISTORIAS CLINICAS

Caso 1.—Niño de nueve años.

Diagnóstico.—Esclero queratitis con irido ciclitis crónica, O. I.

Sin antecedentes familiares—Wassermann negativo—Arneth a la izquierda—Pirquet negativo—Dolores en el ojo—Hipotensión ocular—Visión cero—No presenta sintomatología tuberculosa en ninguna parte del organismo—Desnutrición. No mejora con un tratamiento apropiado.

Etiología probable—Tuberculosis.

Enucleación y prescripción de tónicos generales. Estudio histológico del globo ocular. Inclusión en celuidina—Coloración de los cortes por el metodo Van Gieson—Hematoxilina y eosina.

Esclerótica (Fig. 1).—Nódulos situados casi en el límite exterior de la esclerótica, perfectamente limitados y rodeados de terreno normal.

Células gigantes características de trecho en trecho dispuestas irregularmente de gran talla redondeadas dos en el núcleo superior y una en el inferior.

Espesamiento total de la esclerótica producido por distensión de los espacios lacunares, llenos de células de pepueño tamaño, redondas provistas de núcleo, recordando leucocitos emigrados de territorios vasculares inmediatos.

Esta zona de infiltración dá la imagen de un proceso de escleritis sin

apellido, es la verdadera zona productiva, primera fase de la infección tuberculosa.

Focos tuberculosos más antiguos en fase de degeneración caseosa. Existen verdaderos oquedades, pérdidas de sustancia, rellenas de una masa turbia semisólida, sembrada de gran cantidad de granulaciones de naturaleza grasa o protéica.

Es el resultado de un proceso de desintegración celular por desecación del protoplasma y destrucción del núcleo este proceso degenerativo dá origen a las granulaciones que rellenan las cavernas.

La estructura histológica de la esclerótica esencialmente constituida por haces de tejidos conjuntivo de direcciones cruzadas—antero-posteriores—



Fig. 1.—Nódulos tuberculosos situados en el limite exterior de la esclerótica.

transversales—y oblicuo dan a esta capa pobre en elementos basculares grandes condiciones de resistencia a la propagación.

Sin embargo la infección tuberculosa de la esclerótica misma se propaga en otras direcciones hacia el interior buscando la coroides al exterior por el punto de menor resistencia (vasa vorticosa) a la cápsula de Tenon dando origen a las tenonitis tuberculosas (Fig. 2). Hacia adelante por el limbo esclero corneal a la córnea y por su proximidad en esta región a la raíz del iris y cuerpo biliar.

Pero antes de pasar a describir las manifestaciones tuberculosas en la córnea iris coroides y nervio óptico haremos el estudio detallado de un poco tuberculoso.

Células gigantes (Fig. 3).—Se destaca en el centro de la preparación una célula gigante, punto de referencia esencial para el diagnóstico de la infección tuberculosa.

Son de gran talla 40 a 70 micras de forma redonda o poliédrica, poseen numerosos núcleos algunas veces más de 40 se encuentran situados en la perifería formando una corona alrededor del protoplasma.

El número de células gigantes varía mucho, en nuestra preparación es posible distinguir tras casi juntas, en otras zonas aparece una aislada en otras no hay células gigantes.

En la constitución del módulo tuberculoso se encuentran innumerables



Fig. 2.—Propagación de la infección tuberculosa de esclerótica a cápsula de Tenon.

células conectivas fusiformes de núcleo alargado dispuestas irregularmente en capas concéntricas.

Los fascículos conectivos son tanto más escaso cuanto más nos acercamos al centro del foco.

Al lado de estos elementos celulares se encuentran innumerables células pequeñas de núcleo redondo, que recuerdan en un todo leucocitos emigrados.

Existen células cebadas de Ehrlich que tienen como caracter ser mononucleares con granulaciones protoplasmáticas que se tinen en rojo por la tionina.

Como fase anterior de las células gigantes es posible descubrir elementos epitelioides, con púsculos grandes con núcleo grande y generalmente en vías de segmentación.



Fig. 3.—Foco tuberculosa—células gigantes.

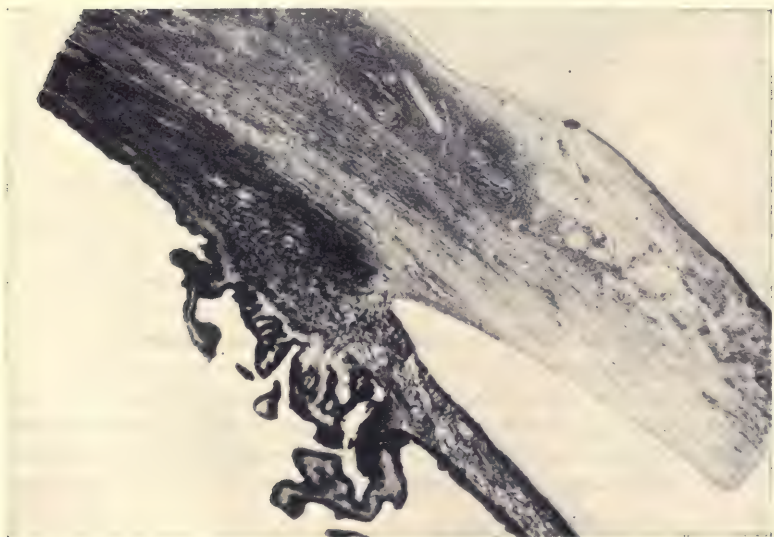


Fig. 4.—Esclero-queratitis tuberculosa.

La propagación de la infección tuberculosa de la esclerótica a córnea en nuestro caso determina una localización poco frecuente que se denomina esclero-queratitis.

En la literatura oftalmológica según la referencia hecha por Morax, en su libro *Pathologie oculaire* 1921 sólo existen publicados tres casos de Wimmerslager y otro de Oreste.

Esclero-queratitis tuberculosa (Fig. 4) las primeras alteraciones aparecen en la proximidad de la inserción de los músculos rectos y aumentan de un modo progresivo hasta ocupar el máximo en la línea esclerocorneal.

Constituye un peligro enorme para la raíz del iris y el cuerpo ciliar aunque la resistencia es grande, en nuestro caso en este punto el iris y ciliar están

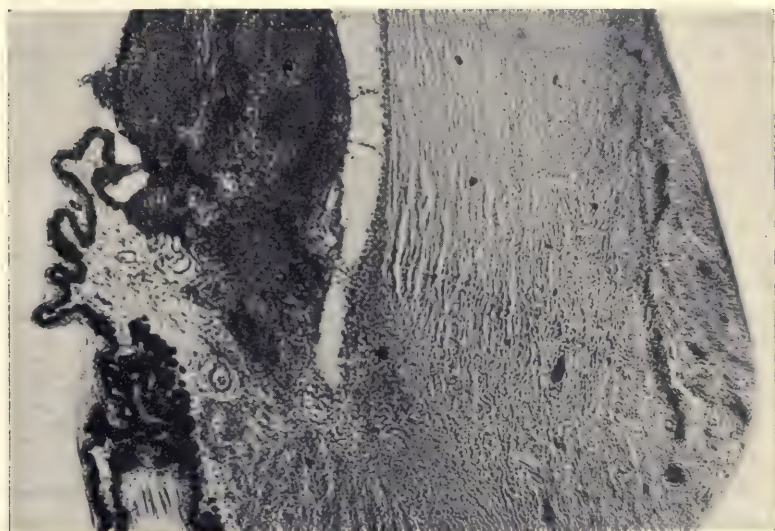


Fig. 5.—Tuberculosis de la raíz del iris.

indemnes, sin embargo en otra zona que vereis en la Fig. 5 podreis apreciar al proceso de propagación a la raíz del iris. La córnea sufre una proliferación bastante marcada de su epitelio anterior ofreciendo un aspecto francamente ondulado.

La sustancia propia de la córnea se modifica y es posible apreciar infiltración leucocitaria en el tercio anterior de las láminas de la córnea, correspondiendo el máximo a la línea esclero corneal, es imposible encontrar células gigantes se confirma el diagnóstico por las lesiones tuberculosas de las otras zonas.

En las láminas anteriores de la córnea existen vasos neoformados colocados muy próximos a la membrana de Bowman sin atravesarla. Alrededor de

estos vasos la infiltración leucocitaria mononuclear es muy marcada existe también en la pared del vaso y adheridos a la túnica interior.

Al nivel de la esclerótica las lesiones están relativamente más marcadas que en la córnea, el espesor del segmento anterior de la esclerótica es por lo menos doble del espesor normal. Esto es debido a una infiltración celular que despegas las láminas de la esclerótica y a una dilatación de los vasos en particular la capa de los vasos superficiales de la esclerótica, a trechos la infiltración forma verdaderos nódulos, estas infiltraciones disminuyen a medida que se acerca a la región ecuatorial. En nuestro caso presenta el iris focos típicos de infección tuberculosa igual que uno de los casos descritos por Wemmerslagen, es posible encontrar de trecho en trecho zonas transparentes que son el resultado de una verdadera condensación del tejido sin sufrir necrosis por coagulación.

Es muy interesante el estudio de la membrana de Descemet aparece ondulada y despegada en algunos puntos de las láminas internas de la córnea.

La capa endotelial presenta en algunos puntos doble pila de células, aparece interrumpida al nivel de la soldadura irido-corneana, por estas rupturas se escapan al ángulo de la cámara anterior en sudados productos del proceso inflamatorio de córnea e iris.

En nuestra preparación existe disminución en profundidad de la cámara anterior por expresamiento del iris. En él existen nódulos tuberculosos agrupados con tendencias de invasión al cuerpo ciliar.

Su localización corresponde a la cara anterior del iris y muy próximo al mismo ángulo irido-corneal.

Miden los nódulos dos m.m. en extensión en el examen vivo aparecen de un color gris amarillento, el color más pálido de la zona central corresponde a la zona de necrosis.

Las alteraciones histológicas más interesantes son:

La destrucción casi total de la capa epitelial anterior, ausencia completa de las criptas de Fuchs, en algunos puntos es posible recordarlas por la ausencia de la membrana basal, dando lugar a verdaderos islotes de leucocitos mononucleares.

La membrana basal anterior aparece ondulada, pero sin romperse vaso de nueva formación se apoyan en ella sin perforarla.

En la sustancia propia del iris constituida principalmente por fibras musculares lisas y estroma conjuntivo se encuentran innumerables células conectivas fusiformes de núcleo alargado y dispuestas en capas concéntricas.

Las granulaciones pigmentarias libres se agrupan irregularmente alrededor del foco tuberculoso y dan la sensación de desorden y enmascaran los detalles finos de estructura.

La membrana basal posterior aparece en muchos sitios rota y sobre ella en grupos irregulares aparecen acumulados las células epiteliales pigmentadas de la capa posterior del iris.

En las arterias se encuentran infiltración leucocitaria en las paredes y muchos de estos elementos adheridos al endotelio. Las arterias ciliares largas y cortas posteriores igual que las ciliares anteriores son la vía de propagación del proceso tuberculoso a cuerpo ciliar y coroides, aunque nuestro caso no hemos de olvidar la propagación directa desde la esclerótica. Focos tuberculosos en coroides (Fig. 6).

Es la membrana del globo ocular que con más frecuencia parece la localización tuberculosa en terreno abonado por lo intrincado de su red vascular.

Se presenta bajo tres aspectos distintos: Tuberculosis difusa, miliar y tuberculoma.

En nuestro caso corresponde al tipo de la tuberculosis difusa, es posible

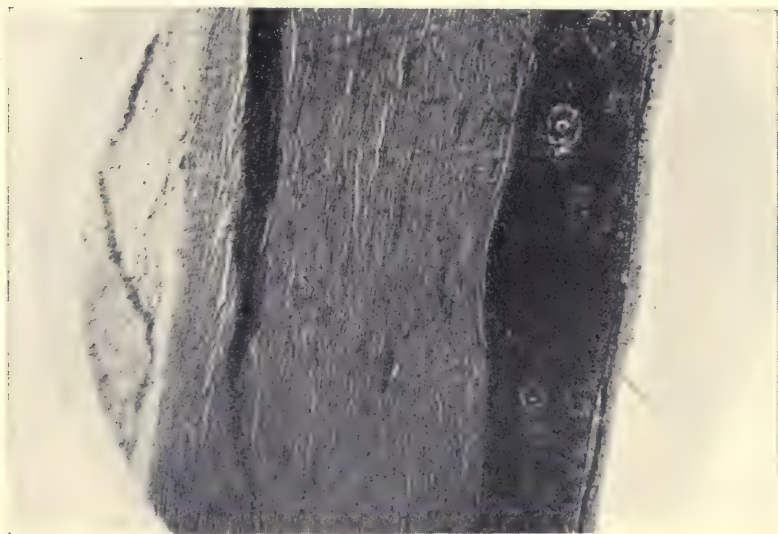


Fig. 6.—Focos tuberculosos de la coroides.

encontrar células epiteliales, células gigantes, infiltración leucocitaria, desaparición del espacio supra-coroides de Schwalbe, es decir de los espacios libres constituidos por un sistema de trabéculas o laminillas conjuntivas que están llenos de leucocitos mono-nucleares, la línea de demarcación de esclerótica a coroides es sinuosa.

El estroma corioideo constituido por fibras de tejido conjuntivo, elásticas finas, musculares lisas, de Muller aparece por los elementos de infiltración dificultando la circulación de arterias y venas por comprensión.

Los capilares de la membrana de Bruch están obliterados.

La membrana vitrea está aumentada de espesor tiene un aspecto fibrilar en conjunto, ha desaparecido la porción lisa que mira la retina.

Tuberculoma de coroides.—La tuberculosis de coroides en esta forma, sólo se puede diagnosticar como tal en los primeros momentos de su desarrollo al aumentar de volumen el tuberculoma produce desprendimiento de retina y glaucoma secundario que hace imposible el diagnóstico con el oftalmoscopio.

La conducta más prudente en estos casos es practicar la nucleación por la posibilidad de la existencia de un sarcoma. Una vez enucleado el ojo el estudio histológico puede aclarar el diagnóstico.

Tuberculosis miliar de nervio óptico.—Esta preparación nos indica la posibilidad de infectarse el ojo de un proceso meníngeo tuberculos o que la meninge se tuberculice por generalización de un proceso ocular (Fig. 7).

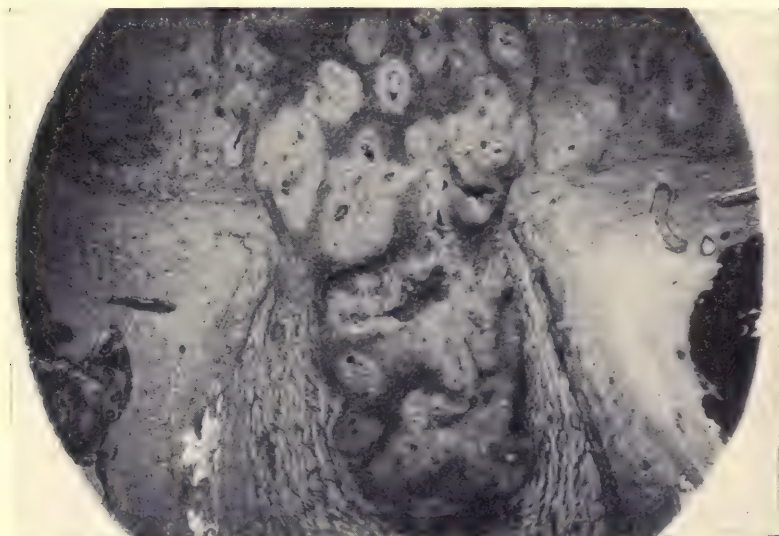


Fig. 7.—Tuberculosis miliar de nervio óptico.

En esta preparación se distingue con toda claridad focos tuberculosos con sus células gigantes. Focos necróticos que da como resultado la destrucción de los elementos nerviosos (cilindro ejes).

CASO 2.—Diagnóstico: Glaucoma secundario—post hemorragia vitrea y en cámara anterior.

Niña de 13 años sufrió un golpe en el ojo derecho hace tres años, desde entonces comienza a perder vista sin ningún dolor, el ojo se le hizo más pequeño, fué diagnosticado de irido-ciclitis traumáticas. Hace más de un año ha perdido totalmente la visión de su ojo derecho.

Antecedentes: El padre murió tuberculoso tiene Wassermann negativo—Pirquet positivo no existe hemofilia—desnutrición—coficiente de Arneith a la izquierda. Padebió de pequeño un tumor blanco de rodilla.

En el momento de esta observación padece intensos dolores glaucoma agudo no tiene perfección luminosa, y ante el temor de una cosa tumoral, cuerpo extraño intraocular o la posibilidad de un proceso crónico tuberculoso enucleamos el ojo como medio profiláctico.

Examen histológico del globo ocular.—Inclusión en celuidina.—Cortes tenidos por ematxilina y eosina.

Las lesiones que hemos encontrado en este globo ocular difieren totalmente de las descritas en el caso anterior.

Son lesiones que denominaremos terciarias desde luego tuberculosas al eliminar la existencia de tumor y cuerpo extraño.

Teniendo presente el tipo degenerativo de las lesiones la reacción de Pirquet positiva antecedentes familiares y personales tuberculosos.

Son cavernas tuberculosas de cuerpo cilia e iris corresponden en enfermo al tipo clínico de hemorragias juveniles en vitrio de origen tuberculoso.

Lesiones de irido-ciclitis tuberculosa y glaucoma secundario a la hemorragia en vitrio y Cámara anterior algo análogo (pérmítidme la comparación) a la hemoptisis del tuberculoso pulmonar terciario.

En tuberculosis pulmonar período carvernario equivale a período terciario es el resultado de la eliminación de los productos de reblandecimiento después de la fase de degeneración caseosa.

Caverna ciliar.—Enorme caverna en pleno cuerpo ciliar llena de sustancias proteicas grasas y sangre (Fig. 8). El músculo ciliar aparece intacto. El canal de Schlemm totalmente obstruido. Los elementos de desintegración celular consecutivos a la degeneración caseosa de los focos tuberculosos no sólo llena la caverna ciliar sino que buscan salida hacia el vitreo.

Existe la destrucción completa de todo el plexo arterio-venoso, la ruptura de estos elementos dan lugar a las hemorragias en vitreo y en cámara anterior (Hemorragias juveniles de origen tuberculoso).

La caverna está limitada por algunos elementos conjuntivos y musculares unidos a lámina y a células pigmentarias.

Como resultado del proceso inflamatorio crónico de ha formado apoyada en el ligamento suspensorio del cristalino una masa que fija la lente que ha sufrido la degeneración cataratosa.

Existe además un estado glaucomatoso secundario, el iris propulsado contra la cara posterior de la córnea ha sufrido también la degeneración cavernaria todo su estroma ha desaparecido, sólo quedan limitando la ojedad fibras conjuntivas y células pigmentarias.

El vaciamiento de la caverna ciliar no sólo tiene lugar hacia el vitreo, sino también puede ocurrir en la cámara anterior, dando lugar a lo que clínicamente se denomina y hipopion y los elementos que lo constituyen dominan productos de esudación hipema, si la sangre entra como factor más importante.

En nuestro caso el color pardo del contenido de la Cámara anterior nos hizo pensar en hipema.

El examen histológico de este globo ocular parece descubrir una forma de tuberculosis crónica que haciendo el estudio comparativo con la tuberculosis pulmonar sería un caso de tercer periodo, terminal o de excavación.

Degeneración esclerótica de la coroides.—En una zona de la coroides donde los tubérculos no han sufrido la evolución fatal de la degeneración caseosa, la toxina tuberculosa ha producido el inquistamiento de las lesiones inflamatorias y ha determinado la esclerosis total.

La coroides ha quedado transformada en una serie de fascículos fibrosos, aparecen algunas cavernarias, es posible distinguir en lugar de alguna célula gigante por la disposición concéntrica de los elementos fibrosos.



Fig. 8.—Enorme caverna en cuerpo ciliar.

¿Esta lesión esclerótica de la coroides nos permitirá pensar en que la localización primitiva de la infección tuberculosa en este ojo ha sido en coroides? Puede ser este proceso esclerótico un mecanismo de curación espontánea.

De todos modos la retina sufre intensos trastornos nutritivos y en temperatura que queda completamente inutilizada.

Los dos casos descritos hasta ahora corresponden a globos oculares con proceso tuberculoso generalizado que termina con la vida del ojo por su destrucción total.

Cuando la lesión se localiza en un sitio determinado produce alteraciones que muchas veces no se imputan a un proceso tuberculoso.

CASO 3.—Ciclitis tuberculosa con luxación de cristalino en la Cámara anterior.

Niña de once años, con lesiones tuberculosas pulmonares—Gram demarcación—Wassermann negativo. Hace tres años sufrió un golpe en el ojo izquierdo es completamente inútil.

Hace una semana ha comenzado a sentir fuertes dolores en el ojo.

Al explorarle nos encontramos con un ojo duro con signos de proceso glaucomatoso bastante intenso con perfección luminosa y con el cristalino luxado en la cámara anterior. Se le practicó la enucleación. Estudio histológico del globo ocular. Inclusión en celuidina—Coloració de los cortes con Hematoxilina y eosina.

Dividido el globo ocular por un corte antero-posterior utilizamos medio ojo para hacer cortes en este sentido y en el otro medio practicamos cortes en el sentido vertical perpendiculares al eje antero-posterior.

Procesos ciliares y zónula de Zinn. Desde la ora serrata hasta el cristalino la membrana hialoides se engruesa y se hace más resistente.

En la masa de este engrosamiento aparece una serie de fibrillas de naturaleza elástica que sirven para fijar el cristalino a la zónula, estos elementos se denominan ligamento suspensorio del cristalino.

Estas fibras se dividen en dos grupos el más abundante vá a fijarse en la cristaloides anterior, otras en menor número ván a insertarse en la cristaloides posterior, algunos autores aceptan un tipo intermedio de fibras muy poco numerosas que se fija en el mismo ecuador del cristalino.

Esta serie de fibras descritas se apoyan por la parte superior en los procesos ciliares plegándose exactamente como ellos, constituye un sistema de elevaciones y depresiones.

Esta descripción hace comprender como un proceso ciclíptico más o menos intenso puede determinar la ruptura o destrucción del ligamento suspensorio de la lente, en un primer periodo se subluxa y en último se luxa, la falta el punto de apoyo y unido a esto los transtornos nutritivos determinan la degeneración cataratosa, disminuye de tamaño y libre el cristalino cae al vítreo o es posible atravesase el agujero pupilar como en nuestro caso y se coloque en la cámara anterior obrando como cuerpo extraño y ser el mecanismo de un proceso glaucomatoso secundario.

Los procesos ciliares han perdido su disposición simétrica, la capa pigmentaria su regularidad, marcándose islotes de pigmento, desaparición completa de los espacios pre-y-post zonulares—No es posible distinguir la disposición radiada de las fibras de ligamento suspensorio.

Cristalino en cámara anterior.—Por delante está en contacto con la cara posterior de la córnea por detrás en relación con la cara anterior del iris.

Los procesos de irido-ciclitis crónica al que corresponde nuestro caso tiene como caracter clínico é histológico la abundancia de exuda-dosplásticos que se depositan en la cara posterior de la córnea, de origen ciliar.

En la formación de estas masas entran leucocitos polinucleares—Células redondas—Hematies—Fibrina.

La córnea aparece íntegra sin ninguna infiltración en las capas profundas.

No existe infiltración ninguna en la membrana de Descemet, ni en su endotelio. El cristalino aparece detrás del iris y en plena degeneración cataratosa. El tipo de catarata en el momento que practicamos la enucleación es cápsula—lenticular.

La cápsula anterior del cristalino se mantiene íntegra. En la capa epitelial existen zonas de peoliteración.

Se descubren de trecho en trecho vacuolas de pequeño tamaño llenas de líquido de Morgagni colocadas debajo de la capa epitelial (Fig. 9).



Fig. 9.—Ciclitis tuberculosa. Luxación de cristalino en la cámara anterior.

Más atrás se encuentra desintegración de las capas de la lente, dejando un gran espacio lleno de sustancia de aspecto granuloso (esferas de Morgagni).

En el iris aparecen zonas de intensa inflamación, focos tuberculosos donde dominan gran cantidad de células gigantes.

Encima de la capa epitelial anterior gran cantidad de exudado a todo lo largo de la superficie del iris (Fig. 10).

En algunos puntos los exudados se reúnen formando masas de gran volumen.

El cristalino por su cara posterior presenta un marcadísimo repliegue de la cristaloides posterior, esto indica una reducción en el volumen de la lente muy marcada, lo que permite que, destruido el

ligamento suspensorio por el proceso ciliático, libre la lente y reducida de tamaño pueda pasar a través del orificio de la pupila y situarse en la cámara anterior.

Qué consideraciones clínicas se deducen del estudio de los casos expuestos?

El valor diagnóstico de la reacción de Pirquet en la tuberculosis ocular varía según el tipo de infección. En el primer caso lesiones tuberculosas primarias sin otras manifestaciones en el resto del organismo el resultado fué negativo.

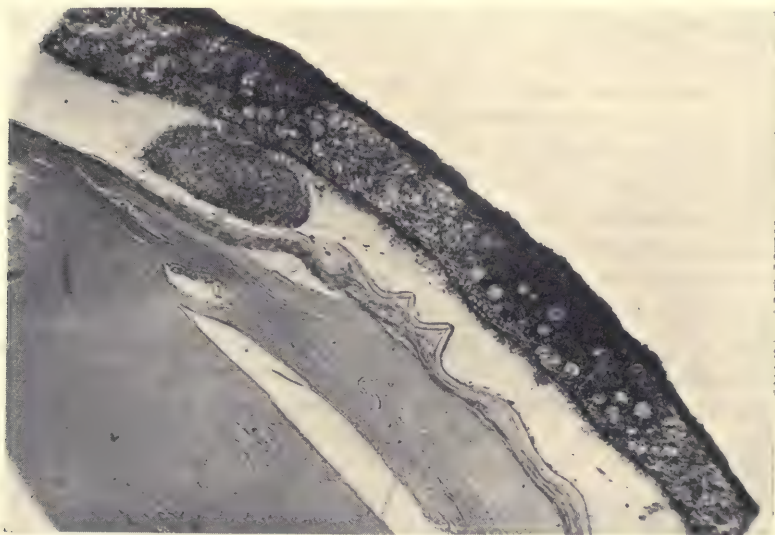


Fig. 10.—Iritis tuberculosa y luxación del cristalino en la cámara anterior.

Los otros dos casos con las lesiones tuberculosas oculares y en otras partes del organismo resultado positivo.

Esto procede indicar que solo tiene valor diagnóstico en los casos de tuberculosis secundaria, pero no debe dejar de hacerse siempre que se sospeche una manifestación primaria un resultado positivo puede ser de gran valor para precisar la etiología.

Nosotros hemos empleado la cuti-reacción y en algunos casos la inyección subcutánea de tuberculosis Koch (O.T.) $\frac{1}{2}$ mg. en niño, este procedimiento nos parece más sensible cuando el estado general sea bueno y la manifestación ocular no sea muy intensa.

No emplearemos nunca la oftalmo-reacción.

Como contra indicaciones para la reacción de Pirquet nosotros tenemos en cuenta:

1. Historia clínica de tuberculosis positiva.
2. Temperaturas frecuentes de 37 grados con 3.
3. Lesiones de corazón.
4. Hemoptisis.
5. Nefritis.
6. Epilepsia.

Hemos comprobado como una lesión primitiva aislada puede determinar por propagación la generalización a todo el ojo del proceso tuberculoso sin propagación al resto del organismo en el momento de la nucleación en el caso 1.

El caso segundo es una forma de tuberculosis cavernaria probablemente consecutiva a localizaciones en otra parte del organismo, este caso nos hace pensar que la tuberculosis ocular puede presentar los mismos períodos que se describe en la tuberculosis pulmonar.

La comparación que hemos hecho entre Hemoptisis y hemorragias en vítreo es perfectamente razonal.

Axenfeld y Stock—1909—Fueron los primeros en fijar el origen tuberculoso de las hemorragias en vítreo, pues tuvo ocasión observarlas en un enfermo con antecedentes familiares y una iritis tuberculosa típica que mejoró por el tratamiento con tuberculina.

Leber, 1913, llamó la atención como origen de las hemorragias en vítreo de los niños la existencia de tubérculos miliares en los vasos de la retina.

Fleischer, 1914, hizo comprobaciones histológicas de alteraciones basculares en vasos de coroides y retina en tuberculosos y esto atribuye el origen de las citadas hemorragias.

Nuestras preparaciones nos hacen admitir que los procesos destructivos de iris ciliar y coroides,—período cavernario que durante su evolución, o en momentos determinandos, el contenido de la cavidad va a vítreo o a cámara anterior simulado una hemorragia olímicamente.

El tercer caso tiene valor en el capítulo de las complicaciones, la más conocida es el desprendimiento de la retina cuando existe localización de focos tuberculosos en la coroides. La luxación de cristalino que no sea de origen traumático no debe olvidarse investigar una posible localización tuberculosa de cuerpo ciliar.

No he de fatigar más vuestra atención, dos palabras respecto al problema terapéutico.

1. El ojo conserva agudeza visual. En toda lesión tuberculosa

ocular ya sea primaria o secundaria debe intentarse a toda costa conservar el ojo ayudando con un tratamiento general intensivo el local apropiado.

2. Cuando la tuberculización del globo ocular sea tan intensa que la función visual esté completamente abolida, y de examen clínico saquemos como consecuencia la pérdida irreparable producida por las lesiones, el ojo debe ser enucleado como medio profiláctico y terapéutico.

DISCUSSION

DR. WILLIAM C. FINNOFF (Denver, Col.): Tuberculosis of the eye undoubtedly exists more often than is commonly supposed. The chief reason for our failure to recognize this, as has been pointed out by Dr. Poyales, is the paucity of material that has been saved for microscopic examination.

Tuberculosis of the eye in children is not seen frequently, but the disease is not rare, and a number of cases have been reported in the literature in recent years. The chronic type of keratoscleritis associated with uveitis is probably the most common form. In many instances the greatest involvement in these cases is in the anterior segment of the globe. In this variety of tuberculosis the subject is frequently robust and apparently in good health, and no positive lesions are found elsewhere in the body by our present known methods of examination, including the *x*-ray. A positive von Pirquet reaction suggests the cause, but is not positively diagnostic as far as the eye condition is concerned. The focal reactions in the eye are positive, and subcutaneous injections of tuberculin (preferably O. T.) are our only means of diagnosis. This, of course, is not to be considered in case of active tuberculosis elsewhere in the body.

The two cases which Dr. Poyales has reported, in which there is a definite history of trauma preceding the tuberculosis of the eye, are of interest because we think of trauma as an underlying factor in the production of tuberculosis of the eye in tuberculous subjects—that trauma makes the soil fertile for the tubercle bacillus. In some experiments we have been carrying out on animals it has been rather striking to find that trauma plays a very small part in the course of tuberculosis. Eyes that are not traumatized run practically the same clinical course as traumatized eyes in animals that have been inoculated with tubercle bacilli.

The classification of tuberculosis of the eye as primary where there is no lesion found elsewhere in the body I believe is often erroneous, unless the tuberculosis exists in a portion of the eye which is either directly or indirectly exposed to the air—other than eyes that have been perforated. The intra-ocular tuberculosis that has been in the past classified as primary, in which the parts were not exposed to the air and not perforated, I believe is secondary in most instances, the primary focus being elsewhere in the body but not discovered. These cases I think should be classified as secondary tuberculosis.

MR. J. GRAY CLEGG (Manchester, England): I want to mention one case. Leprosy, as we are aware, is now being treated with Chaulmoogra oil and

preparations therefrom. I have been using injections of sodium morrhuate in the treatment of ocular tuberculosis, and I am convinced that it is worth trying, for in one case a large number of tubercles rapidly disappeared.

The case I wish to mention is that of a baby who was brought to me by the family doctor with a diagnosis of iritis. Undoubtedly there were signs of that disease, but there was in the iris a small mass exactly resembling a tuberculous nodule. I had the case go to a clinical pathologist, Dr. Arnold Renshaw, who found that the Wassermann reaction was positive in the child and the mother. Under appropriate treatment the condition cleared up. So it is possible for us sometimes to confuse our cases of syphilitic infection of the eye with those of tuberculosis.

DR. IGNACIO BARRAQUER (Barcelona, Spain): No quiero hablar ni del diagnostico ni del tratamiento de todas las formas de la tuberculosis ocular; unicamente quiero llamar vuestra atencion sobre un grupo de enfermos que todos vemos a diario con vitreo turbio, sinequias, descemetitis, ataques de glaucoma secundario, etc., etc., enfermos que diagnosticamos de iridociclitis idiopatica o de la menopausia porque ni el analisis general del enfermo ni los analisis de laboratorio nos ayudan a descubrir la etiologia que pueda orientarnos en el tratamiento. En algunos de estos enfermos el examen microscopico del ojo vivo por medio del cristal de contacto, nos hace descubrir pequenos focos de periflebitis retinal, acumulos de linfocitos en el vitreo, exudados gelatinosos de forma esferica detras del iris que forman sinequias, precipitaciones en la cara posterior de la cornea, grandes, transparentes, hemiesfericas, gelatinosas y entre ellas grupos de forma estrellada de linfocitos. Ellos mejoran y curan rapidamente por medio del tratamiento fotografico y de las tuberculinas.

DR. H. H. STARK (El Paso, Texas): According to the opinion of practitioners in general, all of the civilized race pass through a stage of tuberculosis before the age of puberty, and we are therefore confronted with the problem of each child having had at some time in his life tuberculosis, practically untreated. I do not believe it is well to declare a case negative on a negative von Pirquet. I believe in the specificity of old tuberculin and the focal reactions.

The two cases reported with trauma emphasize the fact that traumatized tissue, whether it be eye or any other tissue of the body, is always subject to tuberculosis. The extent of injury necessary to produce tuberculosis is unknown. It is quite possible that the strain occurring in a case of hypermetropia may produce it.

DR. JOHN E. WEEKS (New York City): We must never lose sight of the fact that tuberculosis and syphilis may exist in the same individual at the same time, and that it is desirable to make the necessary tests to determine the presence or absence of both in all cases of suspected tuberculosis. The diagnosis of a tuberculous lesion in my opinion depends on the production of a local reaction by the introduction of tuberculin into the system, preferably old tuberculin hypodermically. I do not think the von Pirquet test is of any value in cases of this kind except as it may give information regarding

a general tuberculosis. So far as lesions of the eye are concerned, unless the reaction in the eye is occasioned by the introduction of tuberculin into the system, we are not sure that the lesion is tuberculous. The presence of tuberculosis in the system in general frequently cannot be determined by any physical examination, but only by the use of tuberculin. X-ray examinations are often of much value, but are not positive in all cases.

Tuberculosis in my opinion is primary in the eye only in those cases where the anterior portion of the globe is affected—in other words, where the tubercle bacillus can be carried into the tissues of the eye by trauma. In all cases of tuberculosis affecting the posterior part of the eye the condition is secondary.

In regard to the treatment of tuberculosis of the eye, it seems to me that the most efficacious treatment (which should be employed only in those cases where a profound general tuberculosis is not present) is by means of tuberculin given in sufficient dosage to affect the individual just short of a general reaction. Tuberculin therapeutics by means of minute doses given in the treatment of tuberculosis of the eye is in my opinion often absolutely valueless. If the dose is carried out as I have suggested, just short of the point of general reaction, and given every four or five days, as the case may be, the best possible results will be obtained; but the treatment must be continued for some months after the manifestations have apparently subsided to insure against recurrence. We all know that recurrences take place, and these recurrences are more frequent in tuberculosis of the choroid than in tuberculosis of the anterior segment of the globe, although recurrences in corneal and scleral tuberculosis are not infrequent.

MR. E. TREACHER COLLINS (London, England): The question has been raised as to whether the tuberculosis we meet with in the eye is primary or secondary. I am strongly of the opinion that, in a vast majority of cases of tuberculosis of the eye, it is secondary. I think unless the tubercle is inserted into the eye through a perforating injury we may always regard it as a secondary lesion. It seems to me almost inconceivable that tubercle bacilli could gain entrance into the system without any manifestations at the point of entrance, and then circulate until they came to the vessels of the eye before they caused any foci of inflammation. I have some rather definite proof in connection with this matter. Many years ago, when first tuberculin was brought before the profession by Professor Koch, I saw a case similar to those reported by the writer of this paper, *i. e.*, one of tubercle nodules of the eye in a young subject, and we gave an injection of Koch's tuberculin and got a very marked local and general reaction. So marked was the local reaction that the eye became painful and enucleation was performed. Apparently the child was otherwise in good health, and showed no manifestations of tuberculosis that could be detected clinically. A month or so after enucleation of the eye a second injection of Koch's tuberculin was administered, and we again got a very marked general reaction, which definitely showed that there must still have been some other lesion in the body from which the eye probably became infected. It is an important point, I think, in the treatment. If we regard the eye as the primary source of trouble we would be more

inclined to enucleate the eye than if it were secondary. I do not think we ought to enucleate an eye for tuberculosis unless it is thoroughly disorganized as a seeing organ.

I have cut sections of a good number in cases of tuberculosis of the eye in young subjects. I am not quite in agreement with some of the other speakers that the tubercle is most common in the anterior part of the eye. I believe a large number of cases of localized, old choroidal atrophy that we see in children are obsolescent tubercular particles. Dr. Sydney Stephenson brought forward some evidence on this point a few years ago.

A favorite site for the commencement of tuberculosis of the eye is in the lymph-spaces between the ciliary body and the sclera. In several cases I have examined microscopically I found nodules situated externally to the ciliary muscle. That accounts for the early symptoms which we find in these cases of scleritis. Many of the cases begin as patches of scleritis which afterward extend into the cornea, so that we get a sclerosing keratitis. If we follow these cases we find that later the spaces of Fontana become involved in the mass of tubercle, and then tubercles may appear in the iris at the periphery of the anterior chamber; or the tubercular mass may extend through external to the ciliary muscle, invading the ciliary processes and giving rise to diminution of tension. Elastic tissues are exceedingly liable to melt when tubercles come into contact with them. This is seen in the lungs, and also in the tissues of the eye. The capsule of the lens is easily destroyed when the tubercle mass presses upon it, and also Descemet's membrane.

With regard to the treatment of tubercular lesions, I have had some encouraging results from the use of tuberculin. Tubercles in the anterior part of the eye are sometimes seen to dissolve by the use of tuberculin injections. There is great difficulty in fixing the amount of the dose which it is wise to use in these cases. I quite agree with Dr. Weeks that tuberculin should be used only where there is no indication of phthisis. Where the tubercular lesion is localized I do not think we need fear the use of tuberculin. I think it is wiser, however, to begin with small doses and gradually work up, as we do not know how each patient will react to tuberculin. I think large doses are often required, but I prefer to work up gradually. Under such treatment the tubercles may disappear from the eye in a satisfactory way, but if you watch the cases for some years afterward it will be observed that the tubercle is liable to recur.

DR. F. H. VERHOEFF (Boston, Mass.): Personally, I have never seen chronic ocular tuberculosis in an infant, and I notice that this paper does not really refer to infants, but to children. I have seen it in children occasionally. I have a child about three years old under my care now with tuberculosis of the ciliary body and tubercles on the iris and in the filtration angle.

In regard to tuberculosis of the anterior part of the eye, my interpretation of what we find there is somewhat different from that of Mr. Collins. He says that he most commonly finds tubercles between the ciliary body and the sclera. He does not say how they get there, but he evidently assumes that they represent direct metastases through the blood. Some years ago I made

some experiments in an attempt to show just how and where the lesions in anterior tuberculosis occur. Of course, in experimenting with animals, if you introduce virulent tubercle bacilli into the eye the tuberculosis is so violent that it is not comparable to that of human individuals. In order to find out, therefore, just where the tubercle bacilli would naturally lodge I employed dead bacilli. I injected them into the vitreous and the aqueous humor. In these rabbits I found the lesions occurred just where they do in human beings. Inject the bacilli into the vitreous and you will get tubercles on the iris or ciliary body, and you may get tubercles in the vitreous, but the most frequent place is at the filtration angle, and from there they may go back to the subchoroidal space. In most cases the bacilli extend along the veins leading from the filtration angle, producing scleritis, and you may even produce tubercles immediately underneath the conjunctiva of the limbus.

In regard to whether tuberculosis in the human subject is primary or not, of course we naturally presume that it is not primary, but we shall not know certainly as to this until we have a good many autopsies. Patients with chronic ocular tuberculosis, however, practically never die of tuberculosis. Some years ago I did an autopsy in one of these cases and found that the woman had died as the result of tuberculosis of the pituitary body. The primary lesion in that case was a small lesion at the base of the lung, something that could not have been detected during life.

DR. RICHARD KERRY (Montreal, Canada): There is one aspect of tuberculosis of the eye which requires more attention than it has received and that is, that it occurs in two definite clinical forms. The one, characterized by tubercle formation, which tends to progress to panophthalmitis, with destruction of the eyeball; in the other the formation of tubercle is not so obvious, but there is a gummatous exudate which tends to organize, the most usual result being loss of sight with preservation of the eyeball. So far as I am aware the relationship of these two forms to one another has not been established, but urgently demands investigation. There may be some question as to how far the fibrous type is tuberculous, it occurs, however, practically always in members of tuberculous families and there is usually other evidence of tuberculosis in the patient.

As to treatment: We have had a number of unusual results with hypodermic injections of iodine. In my series I have lost only one eyeball, and in that case treatment was withdrawn during the critical period of the disease. The treatment is in no sense specific but acts by raising the resisting power of the body. In a case recently reported by Dr. Louie Stegman, of Battle Creek, Mich., an increase of no less than 40% in leukocytosis, after the fourth dose, was reported, thus demonstrating a rational basis for the action of the drug.

DR. F. POYALES (closing): I wish to thank the members who have taken part in this discussion. I believe that anatomic-pathologic examinations should be done more frequently, and that many times a tubercular origin would be found to explain many secondary ocular lesions.

ENDOPHTHALMITIS PHACOANAPHYLACTICA

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Uhlenhuth,¹ in 1903, showed that rabbits could be immunized by repeated intravenous injections of ox lens protein, so that the blood would give the precipitin reaction with lens protein from any animal source. He further showed that these immunized animals failed to give the reaction with ox serum. In other words, the reaction was tissue specific and not species specific. In 1908 Krauss, Doerr, and Sohma² showed that rabbits could be sensitized by single injections of the lens protein of various animals, so that they would give fatal anaphylactic reactions to the lens protein from any animal source, and they also found that this reaction was likewise tissue specific. In 1909 Uhlenhuth and Haendel³ showed that guinea-pigs could even be sensitized to their own lens protein. These results have since been verified by other observers.⁴

So far as we know, no experiments have hitherto been made relating to the possibility that ocular inflammations may result from injury to the lens in sensitized animals, nor have any observations been recorded that would indicate that certain human individuals are hypersensitive to lens protein.

It is, of course, a well-known fact that after injuries to the lens, traumatic or operative, in some cases inflammatory reaction of greater or less severity follows, while in others no such reaction takes place. Some ophthalmic surgeons have maintained that this inflammatory reaction was always due to infection; others have regarded it as the result of irritation due to lens matter. The latter view was apparently first brought forward by Lagrange and Lacoste⁵ in 1911, who reported eight cases in a series of 100 cataract extractions. Schirmer⁶ previously (1899) had observed a series of cases presenting the same clinical picture in which he had found the aqueous sterile, but which he did not explain on the basis of irritation from lens matter. More recently the view that lens matter may cause intraocular inflammation

has been elaborated by Straub,⁷ who has reported the clinical and microscopic findings in a number of cases of such inflammation which he has termed "Endophthalmia Phakogenetica." He did not consider the possibility that some individuals are more sensitive to lens protein than others, but regarded the inflammation as due to special toxicity on the part of the lens, and maintained that this toxicity increased with age. He did, however, attempt to immunize one patient with such an inflammatory condition by subcutaneous injections of lens protein.

It occurred to one of us that inflammatory reaction following injury to the lens in certain individuals might depend upon hypersensitive-ness to lens protein, acquired or congenital, in these individuals, and its absence to the lack of such hypersensitiveness. We have endeavored to test this hypothesis by clinical and pathologic observations and by animal experimentation.

To ascertain whether or not individuals are hypersensitive to lens protein we have employed the dermal and intradermal tests commonly used for determining sensitiveness to foreign proteins. In all cases pig lenses were used for the tests, and, in some of them, in addition, human lenses obtained at cataract operations and ox lenses. The lenses were obtained under aseptic precautions, and, after removing their capsules, dried in the air for twenty-four to forty-eight hours, and ground to a powder in a mortar with a pestle.

The dermal tests were made by scarifying the skin of the forearm with a v. Pirquet scarifier until serum exuded without blood, and then placing the lens powder on the scarified area and keeping it moist with normal salt solution for about twenty minutes. Controls were made with salt solution only. Positive reactions became observable usually in fifteen to sixty minutes, and consisted in slight hyperemia and elevation of the area. One patient (Case 1) had a very irritable skin, so that the control area also showed considerable reaction, but the area treated with lens protein showed so much greater reaction that there was no doubt that the test was positive.

In the intradermal test we used a 10 per cent. solution by weight of fresh lens in normal saline solution, to which 0.5 per cent. phenol was added, 0.04 c.c. being injected between the layers of the skin. For the control, normal saline solution containing 0.5 per cent. phenol was used. A positive reaction consisted in elevation of the area and hyperemia around it. This was usually not definite in less than two hours, and sometimes not until about twenty-four hours. This test

proved to be the more satisfactory of the two, since it was always positive when the dermal test was positive, and definitely positive when the latter was only slightly so. In some of our more recent cases we have used three graded dilutions in making the intradermal tests.

By means of these tests we have attempted first to ascertain whether patients who have had intraocular inflammation apparently due to rupture of the lens capsule are actually sensitive to lens protein. Naturally, only a few such patients were available to us for this purpose, but, fortunately, these few were exceptionally suitable. Cases 2, 3, and 4 were selected previously on the basis of microscopic examinations, the patients on request returning for the skin tests. The clinical records of these cases, however, were later found to accord with the microscopic findings. In each case the results of the tests were positive.

Following are the cases of this series, twelve in number, in which these tests were made:

CASE 1.—Contusion of right eye with rupture of lens capsule, followed by iridocyclitis. Enucleation and microscopic examination of right eye. Later, iritis and glaucoma in cataractous left eye. Iridectomy. Cataract extraction. Rupture of lens capsule found at operation. Dermal and intradermal tests with lens protein positive.

Thomas S., aged fifty-three, was struck in the right eye by the end of a lever, April, 1919. No perforation of globe. Eye became inflamed, but patient continued to work for four months. First seen at the Massachusetts Charitable Eye and Ear Infirmary, Out-patient Department, February 4, 1920. At this time the lens was opaque, there was posterior synechia below, and a low-grade iritis. Light projection good. Patient advised to have teeth extracted, which he did. April 7, 1920, admitted to wards. Pain in right eye. X-ray examination for foreign body negative. On examination the cornea was clear, there were posterior synechia, slight hypopyon, and marked ciliary congestion. The lens showed a mottled, milky appearance. Light projection was faulty. The left eye showed an immature cataract, reducing vision to 20/100. Complete physical examination made at the Massachusetts General Hospital and Wassermann tests were negative.

June 3, 1920: The condition of the right eye showed no improvement and the eye was removed.

March 20, 1921: Patient readmitted to wards complaining of increasing pain in the left eye, which he had had for three or four weeks. On examination there were found marked ciliary congestion, cornea steamy, anterior

chamber normal depth, pupil contracted, irregular and inactive, lens diffusely opaque. Tension + 1.

March 29: Iridectomy, O. S.

April 7: Tension 24 mm. (Souter.)

April 16: Tension 16 mm. (Souter) (pilocarpin).

April 29: Small hypopyon. X-ray of sinuses negative.

May 8: Considerable congestion. Descemetitis marked.

May 10: Paracentesis.

May 13 to June 10: Course of 13 doses of Cöley's toxins given, with a temperature of 103.2° F. and leukocytosis of 18,000 at one time under this treatment.

June 16: Dr. Verhoeff, concluding that the condition was due to lens matter, attempted an intracapsular extraction. At operation the capsule was found ruptured, so that it could not be grasped with capsule forceps and an ordinary extraction was necessary. As much cortical matter as possible was removed by irrigation.

June 24: Eye white. No anterior chamber. Counts fingers at one foot.

July 14: Operation. Iris first separated from cornea and an iridotomy performed.

July 15: Good anterior chamber. Very little reaction.

July 20: Dermal test with dried pig lens positive.

August 3: Eye quiet, but opening in iris closing. Cornea cloudy throughout, but relatively clear in the center. Vascularization of cornea at periphery. Good anterior chamber. Vision: light perception, projection good.

August 16: Intradermal test with pig lens markedly positive.

August 18: Dermal tests with ox lens and immature human cataract both positive. Also dermal tests with cortical matter from mature cataract and nucleus from same cataract moderately positive.

September 13: Iridotomy.

October 20: Eye has gradually improved. Now white and quiet. Cornea much clearer. Iridotomy opening remains patent. Vision: Counts fingers at six inches.

PATHOLOGIC EXAMINATION

(4079) Right eye: Fixation in 10 per cent. formalin, followed by acid alcohol (Figs. 1 and 2).

The cornea shows very slight infiltration with pus-cells in the posterior layers and beginning vascularization at the periphery, some of the vessels extending for a distance of 2 mm. anterior to the canal of Schlemm. The epithelium and Bowman's membrane are intact. No wound of the cornea is to be found. The tissues of the limbus and the episclera, extending as far back as the equator, are infiltrated with lymphoid and plasma cells. The endothelium of the cornea is absent in places behind the center, but is intact at the periphery. Attached to the endothelium are numerous cells consisting

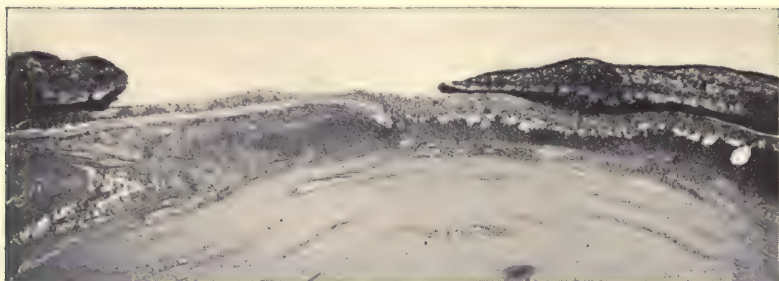


Fig. 1, Case 1.—Showing rupture in lens capsule. The gap is covered by pupillary membrane which is coated on its inner surface by endothelial phagocytes. Beneath this the lens is densely infiltrated with pus-cells, many of which are necrotic. (Photo $\times 25$.)

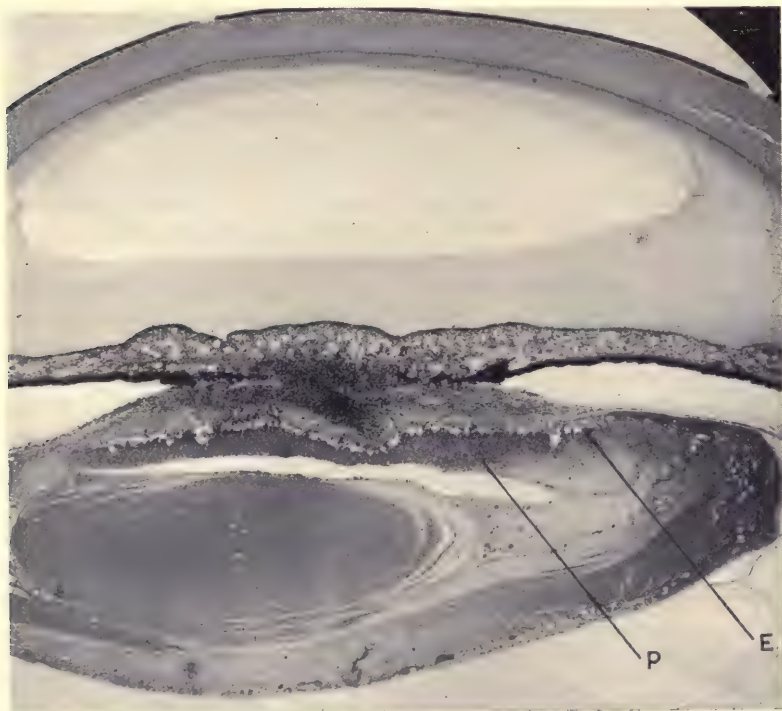


Fig. 2, Case 1.—Showing destruction of pigment layer of iris and infiltrate extending from iris stroma into lens. E, Layer of endothelial phagocytes. P, Massive layer of necrotic pus-cells. Note precipitates on posterior surface of cornea. (Photo $\times 20$.)

chiefly of lymphocytes, and large mononuclear cells, with here and there pus-cells. Over long stretches these cells form a layer one to three times the thickness of Descemet's membrane. Rarely the cells are agglutinated into small clumps. The ligamentum pectinatum is infiltrated with similar cells. The filtration angle is open.

The iris is adherent to the lens by a wide posterior synechia. It is markedly infiltrated with plasma cells, and near the pupil it shows nodular collections of lymphocytes. The pupil is 1.75 mm. in diameter. A pupillary membrane continues from the posterior synechia over the surface of the lens. Attached to the anterior surface of the iris is a thin layer composed chiefly of lymphocytes, which are occasionally agglutinated into small clumps. Some sections show the pigment epithelium of the iris destroyed and the infiltrate extending through the pupillary membrane into the lens. The pupillary membrane is still in active formation and is rich in fibroblasts. It contains also numerous endothelial leukocytes and occasionally giant-cells of the Langhans type, some of which are pigmented. Numerous pigmented cells are present throughout the membrane, but are particularly abundant where the pigment epithelium has been broken through.

The lens capsule shows a defect 2 mm. in diameter. This defect begins on the temporal side of the pupil, most of it being behind the iris. The anterior capsule is swollen in places to about twice its normal thickness and in other places thinned. At the margin of the defect it is swollen and split into two layers, the cleft being invaded by inflammatory cells. The defect is covered by a continuation of the pupillary membrane. This membrane also continues in attenuated form over the capsule to the equator, and in some places around it for a short distance upon the posterior capsule. The endothelial leukocytes and giant-cells are especially abundant over the defect in the capsule. Between the membrane and the lens substance there is a thick layer consisting chiefly of pus-cells, which also extends beneath the capsule on each side. Beneath the capsule most of the pus-cells show marked necrosis. Where they have extended into clefts within the lens substance, necrosis is complete. At one place, a considerable distance from the defect, the capsule is greatly thinned, apparently by the eroding action of the cells attached to its outer surface, which here almost completely perforate it. The lens cortex presents marked cataractous changes throughout, showing numerous spaces and clefts filled with hyaline balls. At the equator there are numerous bladder cells. The lens nucleus, except for a few vacuoles, is practically normal. The pupillary membrane has extended between the capsule and the lens substance, on one side to the equator, on the other side about one-fourth of this distance, destroying the capsular epithelium. The capsular epithelium has not extended beneath the posterior capsule except for a short distance at the periphery, where, in places, it has proliferated and formed a thin capsular cataract.

The ciliary body is comparatively normal. From its surface, particularly from that of the orbiculus ciliaris, is being given off a moderate number of lymphocytes and a large number of mononuclear cells. At the ora serrata cyclitic membranes are in the process of formation. The anterior part of the vitreous contains an increased number of stellate cells and large and small mononuclear cells. The latter are collected in considerable numbers also on the posterior surface of the lens capsule.

The optic disc is definitely swollen, and some of its veins show perivascular infiltration with lymphoid and plasma cells. There is some neuroglia proliferation upon its surface, and the new tissue is infiltrated with lymphoid cells. The optic nerve is apparently normal. Around the disc the retina is edematous, showing dilated spaces in the layer of Henle filled with serum. Some of the retinal vessels here show perivascular infiltration with lymphoid cells. The ganglion-cells are reduced in number anteriorly, but are abundant in the macular region. The choroid is normal.

Sections stained by Gram's method show no bacteria within the globe.

CASE 2.—Spontaneous(?) rupture of lens capsule. Marked iridocyclitis. Enucleation. Microscopic examination of eye. Dermal and intradermal tests with lens protein positive.

Jessie T., aged fifty-eight, came to the Out-patient Department November 24, 1920, complaining of pain in the right eye. Mother was living and blind with cataracts, and one aunt lost both eyes from trouble said to be similar to that of the patient. Twenty years ago patient had iritis, and has had attacks off and on ever since, but never severe. No history of trauma. The onset of the present trouble was a week previous to present date, and consisted of pain and redness of the right eye. On examination the right eye revealed a marked conjunctival and ciliary congestion; the cornea was clear, iris congested, and pupil blocked by a membrane, no increase in tension; light projection good; convergent squint. The left eye was negative as regards disease. Vision O. S. = 20/20. Patient was admitted to the hospital. January 5, 1921, no permanent improvement having taken place in the right eye, and the globe being soft, it was removed.

On October 10, 1921, intradermal and dermal tests with pig lens protein were made on the patient and were markedly positive.

PATHOLOGIC EXAMINATION

(4187) Fixation in 10 per cent. formalin, followed by acid alcohol.

The epithelium of the cornea is intact at the periphery. There is an area 4 mm. in diameter in the center which is denuded of epithelium. Bowman's membrane is everywhere intact, but markedly altered in the denuded area. The stroma is moderately infiltrated with pus-cells in the center and slightly so in the periphery. Vessels are invading the periphery, extending a distance of 2 mm. anterior to the canal of Schlemm. Descemet's membrane is intact

but swollen. The endothelium is intact. There is a peripheral anterior synechia on one side which extends for a distance of 3 mm. Attached to the remainder of the endothelium are numerous cells, consisting chiefly of lymphocytes and plasma cells, with a few large mononuclear and pus-cells. Rarely the cells are agglutinated into clumps. On the other side the filtration angle is free, but the ligamentum pectinatum is sclerosed and infiltrated with lymphocytes, plasma cells, and large mononuclears. The tissues of the limbus and episcleral tissue extending back for a distance of 3 mm. are infiltrated with lymphoid and plasma cells. No wound of the cornea is found.

The iris is adherent to the lens by a large posterior synechia. The portion which is adherent to the posterior surface of the cornea retains a relatively normal structure for a distance of 2 mm. nearest the angle, but is atrophic and infiltrated with plasma cells. It then becomes disorganized and incorporated in a mass of dense connective tissue which forms a pupillary membrane and extends into the lens. Within this mass the sphincter muscle may be seen in the center of a large lymphocytic nodule. The mass is markedly infiltrated with plasma cells, especially around the lymphocytic nodule, and also contains numerous pigmented cells. On the other side the iris is atrophic and markedly infiltrated with plasma cells, and near the pupil it shows a large, nodular collection of lymphocytes. The dense pupillary membrane continues over the posterior surface of the iris to the ciliary body, fuses the iris to the lens, and on one side extends as far as the ora serrata. The anterior capsule is swollen to two or three times its normal thickness in places, while in others it is thinned. At about the center it shows a gap 2 mm. wide through which the connective tissue from the pupillary membrane invades the lens substance. The lens epithelium is proliferated in places and completely destroyed in others. At the anterior and posterior poles the lens has undergone degeneration and is infiltrated with lime salts, but is here practically free from cells. The dense connective tissue invading one side of the lens has walled off a portion of cataractous lens matter at the equator. The other side of the lens is also invaded by connective tissue to some extent, and shows deposits of lime salts. In the central portion of the lens is a somewhat oval mass of lens substance, probably the nucleus, measuring $3\frac{1}{4}$ by $3\frac{1}{2}$ mm. in size. This is thickly surrounded by endothelial phagocytes and giant-cells, a few of which are of the Langhans type, and many pus-cells. The latter cells have invaded long clefts in the lens substance and are here undergoing necrosis. The cyclitic membrane on one side sends a thin layer of fibrous tissue over the posterior surface of the lens. The ciliary body shows very slight cellular infiltration, the active process here evidently having subsided.

The stellate cells of the vitreous are markedly increased in number, and the anterior vitreous contains also numerous round, swollen cells, with eccentrically placed nuclei.

The optic disc shows a moderate papilledema and proliferation of its super-

ficial neuroglia. The optic nerve appears normal. The retina shows central senile retinitis, with marked proliferative and degenerative changes of the pigment epithelium associated with destruction of the rods and cones and replacement of the outer layers of the retina by proliferated neuroglia. In addition, the retina shows several large cystic spaces, an especially large one filled with serum occupying the fovea. The retinal ganglion-cells are present, especially in the fundus.

The choroid appears normal.

Sections stained by Verhoeff's modified Gram's stain show no bacteria within the globe.

CASE 3.—Spontaneous rupture of capsule of cataractous lens. Marked iritis with descemetitis and hypopyon. Enucleation. Microscopic examination of eye. Dermal and intradermal tests with lens protein positive.

Julia V., aged seventy-two, was admitted April 27, 1917, complaining of pain in the left eye, which had its onset four months previously, a month after a successful cataract operation by Dr.—— on the other eye. It began with itching and burning and has gradually grown worse. No history of bodily trauma or injury to eye.

Examination of the left eye at this time revealed moderate congestion of the conjunctiva. Cornea dull, with deposits on the posterior surface. Iris congested. Pupil small, irregular, and did not react to light. Exudate in pupil. No fundus reflex. Light projection good. Diagnosis: Uveitis O. S. The right eye presented the picture of a successful cataract extraction. Vision with correction, 20/40. A complete physical examination at the Massachusetts General Hospital was negative. Patient given 1 per cent. atropin sulphate and hot fomentations.

May 1, 1917: Tension O. D. 18 mm.; O. S. 38 mm. Condition about the same.

May 4, 1917: Pupil moderately dilated. Still has marked descemetitis, otherwise eye quiet and pain has subsided.

May 9, 1917: Pupil dilated and regular. Descemetitis still present. Exudate in pupil. Tension slightly elevated. Very slight congestion. No pain. Patient discharged.

July 3, 1917: Patient readmitted to wards. Had been having very severe pain in O. S. for a month. Examination of O. D. at this time presented same condition as on discharge. O. S. lids slightly swollen. Conjunctiva congested, especially around the cornea. Cornea is roughened and infiltrated. Pupil contracted and filled with exudate. Eye very tender to touch. Anterior chamber shallow. Tension ++. Vision nil.

General examination at Massachusetts General Hospital negative, except for a few râles at base of lungs.

Enucleation.

October 19, 1921: Dermal test with pig lens protein positive. Intradermal

test with pig lens protein doubtful after one and one-half hours, markedly positive after twenty-four hours.

PATHOLOGIC EXAMINATION

(3325) Fixation in 10 per cent. formalin.

The tissues of the limbus and episcleral tissue extending back for a distance of $1\frac{1}{2}$ mm., are moderately infiltrated with lymphocytes and a few plasma cells. The cornea has an area 2 mm. in diameter denuded of epithelium. Bowman's membrane is intact and apparently normal. There is very slight infiltration of the stroma with leukocytes and beginning vascularization, some of the vessels extending $1\frac{1}{2}$ mm. anterior to Schlemm's canal. Above there is a peripheral anterior synechia 2 mm. wide. Below, the filtration angle is open. Descemet's membrane is intact. The endothelium is intact in the upper portion, but destroyed in the lower.

The lower part of the posterior surface of the cornea is coated with a membrane partly organized and rich in cells. This membrane begins as a thin layer 5 mm. from the filtration angle above, and gradually becomes thicker below, reaching a thickness of $1/16$ mm. It contains many fibroblasts evidently derived from the corneal endothelium, numerous pus-cells, and large and small mononuclear cells. The pus-cells are abundant next to Descemet's membrane. The posterior surface of the upper part of the cornea is practically free from cells for a considerable distance; then clumps of mononuclear cells appear, which increase in size and number as the membrane just described is approached.

The lower filtration angle is filled with a hypopyon $2\frac{1}{4}$ mm. high, composed of serum, fibrin, and cells. The cells are mostly pus-cells, plasma cells, and endothelial leukocytes. The endothelial leukocytes are intensely phagocytic, being engorged with chromatin fragments. The hypopyon also contains a moderate amount of blood.

The iris is adherent to the lens by a large posterior synechia on both sides. The pupil is $1\frac{1}{2}$ mm. wide, and filled with a membrane rich in cells, many of which contain pigment. This membrane continues between the iris and lens capsule for a distance of $2\frac{1}{2}$ mm. in places. The anterior surface of the iris above is relatively free from cells. Below it is coated with numerous cells, consisting chiefly of large and small lymphocytes, pus-cells, and endothelial phagocytes. The iris stroma is markedly infiltrated with plasma cells, especially posteriorly.

The lens capsule shows a defect of $2\frac{1}{2}$ mm. on the anterior surface. The free ends of the capsule are split, and the clefts invaded by endothelial leukocytes and a few pus-cells. On each side of the defect the lens capsule is swollen. The lens epithelium is markedly proliferated at the edges of the defect, and is apparently giving rise to a few phagocytic cells. Otherwise the epithelium appears normal. Along the inner surface of the pupillary membrane there are

numerous endothelial leukocytes. In places these have formed syncytial masses and giant-cells. Beneath these, directly bordering on and invading the lens substance, there is a layer of cells, $\frac{1}{3}$ mm. in thickness. These cells are almost exclusively pus-cells, many of which have undergone necrosis. In places the pus-cells have extended into deep clefts within the lens substance, and here are completely necrotic. The remainder of the lens cortex is cataractous, but the nucleus appears normal.

The ciliary body is very slightly affected, being only sparsely infiltrated with plasma cells in the anterior portion. From the surface of the pars plana below there is a moderate exudation of lymphocytes and plasma cells.

The optic nerve and disc are apparently normal. Some of the vessels in the retina show slight perivascular infiltration with lymphocytes. Otherwise the retina appears normal. The choroid is apparently normal.

Sections stained by Verhoeff's modified Gram stain show no bacteria within the globe.

CASE 4.—Perforating wound of cornea, iris, and lens. Removal of foreign body by magnet. Iridocyclitis. Enucleation. Microscopic examination of eye. Intradermal test with lens protein positive.

Henry Q., aged thirty-nine, was admitted to the Massachusetts Charitable Eye and Ear Infirmary May 21, 1919. A few hours before, while hammering a pipe, a piece of steel flew and struck him in the left eye. His history was otherwise negative. Examination at the time of admission was negative except for the injury to the left eye. The eyelids of the left eye were slightly swollen and congested. The bulbar conjunctiva was also congested. The cornea showed a large horizontal perforating wound at the lower edge of the pupillary area. The iris and lens showed a triangular opening on the temporal side a little above the corneal wound. Pupil reacted to light. Fundus was seen with difficulty. Anterior chamber of normal depth. Transillumination was negative.

X-ray examination showed a foreign body localized 11 mm. back, 4 mm. temporal side, $4\frac{1}{2}$ mm. below, size $1 \times \frac{1}{2}$ mm.

May 22, 1919: Dr. Derby removed the foreign body through the wound in the cornea with the magnet.

May 23: Anterior chamber reformed. Eye very red.

May 26: Eye about the same; not much pain.

May 31: Marked iritis. Lens swollen and cataractous. Pupil fairly well dilated. Pain at times.

June 7: Not much pain. Eye still congested.

June 18: Slightly better. No pain. Not so much congestion.

June 26: Eye congested; pain at times.

July 1: Enucleation.

October 24, 1921: Intradermal test with pigs lens protein is positive. The dermal test is doubtful.

PATHOLOGIC EXAMINATION

(3744) Fixation in 10 per cent. formalin.

The cornea shows a large horizontal linear healed perforating wound just below the center. The corneal epithelium is everywhere intact, but shows evidence of bullous keratitis in areas. Bowman's membrane is intact except at the site of the wound. The stroma is practically free from cellular infiltration. The edges of the wound are closely approximated, and the gap filled with connective tissue rich in cells. The contiguous stroma shows very little evidence of past reaction. At the periphery, all around, there are a few new-formed blood-vessels extending for a distance of $1\frac{1}{2}$ mm. anterior to the canal of Schlemm. The limbus and episcleral tissues extending as far back as the equator in places are markedly infiltrated with lymphoid and plasma cells. Descemet's membrane is intact, but absent over short stretches, and is everywhere coated with large mononuclear cells. Often these are agglutinated into large clumps. Among these also are a moderate number of pus-cells and an occasional eosinophile. Attached to the endothelium are a number of small masses of altered lens matter which have been invaded and surrounded by large mononuclear cells and a few cells resembling fibroblasts. The anterior chamber is filled with serum containing pus-cells, lymphocytes, and occasionally an eosinophile. The cells become more numerous as the lower angle is approached, where they have accumulated in the form of a hypopyon $1\frac{3}{4}$ mm. high. The hypopyon contains also a considerable amount of broken-down lens matter. The ligamentum pectinatum is markedly infiltrated with similar cells.

Some sections show beginning adhesion of the iris root to the ligamentum pectinatum. The iris is also adherent to the anterior surface of the lens capsule by a wide posterior synechia. The anterior surface of the iris is covered by a layer of leukocytes consisting of lymphocytes, large mononuclears, and pus-cells. Some of the large mononuclears are agglutinated into clumps. The lymphocytes and pus-cells are relatively more abundant than on the posterior surface of the cornea. The stroma of the iris is markedly infiltrated with lymphocytes and plasma cells, the latter predominating, and also shows nodular collections of lymphocytes.

The pupil is filled with a dense vascularized fibrous mass rich in fixed cells, forming a cone which is continuous with the tissue filling the corneal wound. This mass is infiltrated with pus, lymphoid and endothelial cells, some of the latter containing pigment. The center of this cone contains a mass of broken-down lens matter invaded and surrounded by pus-cells and endothelial phagocytes.

The lens capsule shows a defect of $1\frac{1}{4}$ mm. in the lower half of the anterior surface, and another of $\frac{1}{2}$ mm. near the equator on the posterior surface of the same side. Where the capsule is intact, it is adherent to the iris and ciliary

processes. The anterior capsular epithelium is destroyed in places. On one side the pupillary margin of the iris continues into a thick mass of connective tissue. On the other side it is free from this mass, but adherent to the lens capsule. Through the gap in the lens capsule the connective tissue has extended into the lens substance to a depth of $\frac{1}{2}$ mm., and spread beneath the capsule on one side for a distance of about 1 mm. Beneath the connective tissue the lens shows marked cataractous changes, is breaking up into hyaline balls, and shows numerous clefts. It is being invaded by numerous endothelial phagocytes, which in some places are so abundant that they have formed a syncytium. The lens substance in this area is also markedly infiltrated with pus-cells many of which have undergone necrosis. The posterior cortex shows cataractous changes, and beneath the posterior capsule is invaded by endothelial phagocytes which have extended from the gap in the posterior capsule. From the surface of the ciliary body below there arises a thick cyclitic membrane, still in active formation, which extends up to and through the gap in the posterior capsule. Within this membrane, near the gap, are large masses of disintegrated lens material.

The ciliary body is markedly infiltrated with plasma and lymphoid cells. Below it is, in addition, highly edematous. The surface, above, is covered with an exudate containing endothelial and pus-cells, about which a cyclitic membrane is being formed. This membrane extends as far back as the ora serrata, and is insignificant in comparison with that over the lower part of the ciliary body already mentioned.

The vitreous body contains a small disintegrating foreign body about 1/10 mm. in diameter, evidently left behind when the main foreign body was removed. This is inclosed by endothelial cells which thus form a small foreign body tubercle free in the vitreous.

The optic disc shows moderated papilledema with fairly well-marked perivascular infiltration of the vessels. In the center beneath the surface is an area infiltrated with lymphoid and plasma cells. There is also some proliferation of the surface neuroglia cells. The optic nerve appears normal.

The retina shows marked perivascular infiltration with plasma cells and lymphocytes. Attached to the surface of the retina there are many plasma cells, lymphocytes, and large mononuclear cells. Immediately beneath the internal surface there are numerous small foci of neuroglia proliferation. Some of these contain a slight amount of iron pigment and some of them project in nodular form into the vitreous. The ganglion-cells are everywhere greatly diminished in number except in the macular region, where they are still abundant.

The choroid is congested but free from infiltration except near the ora serrata, where it shows perivascular infiltration.

Sections stained by Verhoeff's modified Gram stain show no bacteria within the globe.

CASE 5.—Extraction of mature senile cataract, followed by iritis and glaucoma. Enucleation. Microscopic examination of eye. Intradermal tests with lens protein positive.

This was a private case of Dr. Geo. F. Worcester's, to whom we are indebted for the clinical notes. The patient was seen by Dr. Verhoeff in consultation December 9, 1921, at which time the intradermal tests were made.

Mrs. H. A. S., aged seventy-three, mature cataract O. S. immature, O. D. June 21, 1921: Preliminary iridectomy, O. S.

July 30: Cataract extraction, O. S. Some soft lens matter left.

Anterior chamber found reformed at first dressing. Reaction slight. No prolapse of iris.

August 10: Anterior chamber obliterated.

August 27: Iritis with descemetitis. Edema and congestion of lids (atropin irritation).

September 15: During past month, anterior chamber has reformed and precipitates on posterior surface of cornea have diminished. The eye is now quiet.

September 19: O. S.: Anterior chamber obliterated. Eye very much congested. Tension 55 mm. O. D.: Deep anterior chamber. Pupil dilated. Slight ciliary congestion. (Subsequent events showed this was an attack of glaucoma, O. D.) Enucleation O. S.

September 24: O. D.: Quiet. V. O. D. = 20/40.

December 6: O. D.: Congested, cornea hazy, anterior chamber deep. No posterior synechia. No descemetitis. Tension 25 mm. V. O. D. = 20/40. Atropin ordered.

December 8: V. O. D. = 20/100.

December 9: Patient seen by Dr. Verhoeff, who made diagnosis of sub-acute glaucoma and atropin irritation O. D. Eye slightly congested, cornea steamy. Pupil widely dilated. No posterior synechia or descemetitis. Tension 36 mm. (Souter). V. O. D. = 20/200. Skin of both eyelids of right eye edematous and hyperemic. Intradermal tests with pig lens protein made in three dilutions by Dr. LeMoine.

December 10: Dr. Worcester reports all intradermal tests positive.

PATHOLOGIC EXAMINATION

(4319) Fixation in 10 per cent. formalin.

The cornea is slightly infiltrated with pus-cells and shows a few vessels extending into it for a considerable distance from the periphery. The tissue of the limbus is densely infiltrated with plasma and lymphoid cells. The wound is firmly healed everywhere except directly above. Here the anterior lip of the wound overrides the posterior, and the two lips are united by a thick mass of granulation tissue which is densely infiltrated with lymphoid and plasma cells and also contains numerous eosinophiles. On each side the site of the wound is

infiltrated with plasma cells. There is a peripheral anterior synechia, about 2 mm. wide all around, but the iris is nowhere incarcerated in the wound. Attached to the posterior surface of the cornea there is a thick layer composed of large and small lymphocytes which is being organized by the corneal endothelium. There are also, here and there, adherent to the posterior surface, small particles of lens matter containing pus-cells.

The iris is densely infiltrated with plasma cells. From the pupillary margin there arises a thick pupillary membrane which also continues behind the iris, fusing the latter to the lens capsule. It is continuous with the iris stroma through breaks in the pigment layer. This membrane is rich in epithelioid cells and contains a number of giant-cells. Within it there are also foci of pus-cells, evidently due to lens matter which has absorbed.

Within the collapsed lens capsule only a small amount of lens matter remains. In places where it is exposed it is being invaded by pus-cells.

The ciliary body is free from infiltration, and from its surface there is practically no cellular exudation. The ciliary processes are dragged forward and compressed. The choroid is normal. The retina is also normal, except for slight edema of the macula and perivascular infiltration with lymphoid and plasma cells near the disc. The optic disc is not swollen or cupped, but shows considerable perivascular infiltration with lymphoid and plasma cells. The nerve stem shows slight infiltration with lymphoid cells, but otherwise appears normal.

CASE 6.—Combined extraction of immature cataract, with early discission followed by severe iritis and attacks of increased tension. Dermal tests with lens protein positive.

Fred H., aged fifty-five, operated upon for immature cataract, right eye, by Dr. — on September 14, 1920. Five weeks later, discission performed. Following this the eye was greatly irritated and there were attacks of increased tension. Seen by Dr. Verhoeff in consultation on November 17, 1920. At this time the right eye showed moderate congestion but considerable photophobia. Apparently only a small amount of cortical matter remained unabsorbed. The pupil was contracted and drawn up and the iris was adherent to the lens capsule. There was one large white precipitate on the back of the cornea. The tension was 2 mm. (Souter). In view of the history of attacks of increased tension, 2 per cent. solution of pilocarpin nitrate was prescribed. The left eye showed an immature cataract.

May 25, 1921: The patient was again seen by Dr. Verhoeff. Right eye was free from congestion. Pupil largely obstructed by a thick capsule. V. O. D. = 20/70.

May 31: Discission by Dr. Verhoeff. Failing to get a good opening, the incision was enlarged and a large portion of the capsule pulled out by forceps and the iris sphincter cut below. Considerable hemorrhage into the anterior chamber resulted.

July 6: A considerable amount of blood remained in the pupillary area. V. = 20/200 with glasses.

July 7: Operation by Dr. Verhoeff. Iridectomy and capsulotomy. Good opening obtained.

August 2: Dermal tests with pig and ox lens positive.

August 6: Dermal test with human immature cataract positive.

September 27: V. with + 9.50 S. \ominus + 4.00 c. ax. 170 = 20/40. An intracapsular extraction was performed upon the left eye by Dr. Verhoeff. This was not followed by iritis or glaucoma.

CASE 7.—Combined cataract extraction with considerable retention of cortical matter, followed by severe iritis, descemetitis, and infiltration of the cornea. Dermal and intradermal tests with lens protein positive.

Georgiana M., aged sixty-four, came to the Out-patient Department of the Massachusetts Charitable Eye and Ear Infirmary March 3, 1921, complaining of progressively failing vision, which began one and one-half years previously. Six weeks before admission she had a slight attack of inflammation in the left eye, possibly iritis. Otherwise no history of intraocular inflammation in either eye could be obtained.

The examination revealed nothing of importance except double immature cataracts. Both pupils reacted to light. The left eye showed no posterior synechia or pigment on the lens, but the right eye showed one small posterior synechia. Light projection good O. U. On March 5, 1921, Dr. Cheney did a combined cataract extraction on the left eye. Considerable cortical matter was left. The eye was quiet and apparently the cortical matter was absorbing slowly until March 10, when the eye began to be slightly congested. The inflammation increased so that by March 17 the patient had edema of the lids, congestion of the iris, and deposits on the posterior surface of the cornea. Diagnosis: iridocyclitis. On the 23d the patient developed hypopyon. On the 31st the eye was soft with a marked hypopyon. From that time on the patient gradually improved and was discharged from the hospital April 18. At this time the eye was white and quiet. The pupil was drawn up and filled with a dense membrane. The upper half of the cornea showed mottled opacities. Projection was good.

The patient was readmitted to the wards on August 2, 1921. At that time both eyes presented very much the same condition as on discharge except that posterior synechiæ were noted in the right eye. Dermal and intradermal tests with pig lens protein were positive. On August 5 Dr. Verhoeff did a combined intracapsular cataract extraction on the right eye. The patient made an uneventful recovery and was ready to leave the hospital on August 19. At this time a discission and iridotomy was made on the left eye. Following this operation the patient had a marked reaction in the eye.

CASE 8.—Extraction of mature senile cataract by capsulotomy method,

with retention of cortical matter within capsule. Moderate iritis. Dermal tests with lens protein positive.

Mary M., aged sixty-four, was admitted to the Massachusetts Charitable Eye and Ear Infirmary August 31, 1921, complaining of progressively failing vision which began five years ago. Examination at this time was negative except for uncomplicated mature cataracts O. U. Tension was normal. Projection good. V. O. D. = 20/200; V. O. S. = 20/200.

September 1, 1921: Dr. Cheney did a combined cataract extraction O. D. Some cortical matter was left.

September 2: Dermal tests with ox and pig lens protein positive.

September 3: Eye white. Good anterior chamber. Considerable cortical matter. Pupil does not dilate well. Cornea shows striped keratitis.

September 6: Slight chemosis, but not much congestion. No pain.

September 13: Iritis with pain, redness, and slight chemosis.

September 14: Dermal tests with ox and pig lens protein positive.

September 16: Chemosis still present, but iris seems clearer. Less pain.

September 19: Eye much whiter. No pain. Cortical matter protected by the capsule.

September 22: Eye much whiter. Patient discharged. V. O. D. + 10.00 = 5/200. V. O. S. = 2/200.

CASE 9.—Old injury of left eye with iritis, probably due to lens matter. Ten years later, cataract extraction by capsulotomy method right eye, followed by iritis. Later, discission, right eye, followed by iritis. Intradermal test with lens protein positive.

John B., aged seventy-three, was admitted to the Massachusetts Charitable Eye and Ear Infirmary June 20, 1920, with the following history: Ten years ago had an injury to the left eye which caused loss of vision of that eye, but never had much pain following the injury. In September, 1914, patient came to the Out-patient Department complaining of loss of vision O. U. The examination at that time revealed O. D. Tension 34 mm. V. = 20/40. Field of vision contracted about 10°. Disc not cupped. O. S. Tension 50 mm. V. = light projection faulty. The left lens was cataractous, the pupil dilated, irregular, bound down to the lens capsule, and the iris discolored. There was considerable cortical matter left in the capsule, but none free. In June, 1919, vision was failing. V. O. D. = counts fingers at one foot. An immature cataract O. D. was noticed. Tension normal O. U.

At the time of admission the examination revealed the following: O. D.: Nearly mature cataract, light projection good. Tension +. Congestion of lids. O. S.: No change since first examination. Examination at the Massachusetts General Hospital was negative.

June 23, 1920: Preliminary iridectomy O. D.

The patient made an uneventful recovery and left the hospital with a tension of 20 mm. O. D.

October 10, 1920: Cataract extraction O. D. by capsulotomy method by Dr. Lowell.

October 21: Anterior chamber reformed. Complains of pain O. D., especially at night. Eye congested.

November 5: Iris assuming a greenish color and patient has much pain. Eye congested. Pupillary opening drawn far up.

November 29: Eye beginning to quiet down and pain not so severe. Examination at Massachusetts General Hospital was negative.

December 8: Continues to improve.

December 29: Good anterior chamber. Iris markings distinct. No pain. Counts fingers at one foot.

January 3, 1921: Discharged. Eye practically white and quiet.

May 21: Condition about the same as upon discharge.

May 23: Dissection O. D.

May 26: Patient has much pain. Iris much inflamed. Patient referred to Massachusetts General Hospital for a general examination to locate cause of iritis. Examination was negative.

June 26: Referred to the Massachusetts General Hospital, where all lower teeth were extracted.

July 15: Eye whitening. Pupil contracted.

July 18: Discharged.

October 25: Intradermal test with pig lens protein positive.

CASE 10.—Senile immature cataract. Intracapsular extraction with rupture of capsule and retention of some lens matter in anterior chamber, followed by iritis. Intradermal test with lens protein positive.

Mr. S., aged sixty-four, was admitted November 1, 1921, with bilateral immature senile cataracts. V. O. D. = shadows. O. S. = 20/50. No history of any inflammatory condition of either eye.

November 2, 1921: Combined intracapsular extraction with capsule forceps right eye, by Dr. Verhoeff. Rupture of the capsule occurred just as the cataract was being delivered. A moderate amount of cortical matter escaped into the anterior chamber and was allowed to remain there. The entire capsule, however, came away.

November 4: Anterior chamber reformed. Cortical matter about 4 mm. in diameter, adherent to cornea, not in contact with iris.

November 8: Intradermal test with pig lens protein positive.

November 12: There has been a moderate amount of pain, especially at night. The eye is markedly congested. The pupil is only half dilated under atropin. The cortical matter is half absorbed; a small amount of it lies in the lower angle in contact with the iris.

November 14: Pupil more contracted. Eye markedly congested. Moderate amount of pain. The iris below, where the cortical matter lies upon it, is congested and coated with a slight amount of exudate.

November 18: Pain and congestion more marked. Two pieces of cortical matter are adherent to the anterior surface of the iris, one below and one in the lower nasal quadrant. Iris much swollen in the vicinity of each piece of cortical matter and coated with a slight amount of exudate. Eyelids congested and somewhat swollen—atropin irritation? Hyoscin substituted for atropin.

November 23: Lens matter all absorbed except for a small piece adherent to the hyaloid membrane and not in contact with the iris. Congestion much less, no pain. V. O. D. with correction = 20/40.

December 8: O. D. still moderately congested. The iris shows a number of posterior synechiae. A small bit of lens matter remains adherent to the hyaloid membrane.

December 22: O. D. practically free from congestion. The former site of the lens matter on the hyaloid membrane appears as a slightly pigmented spot. V. O. D. with sph. + 12.5 \odot + 1 cyl. ax. 155° = 20/30.

CASE 11.—Immature cataract. Attempted intracapsular extraction. Rupture of capsule and retention of cortical matter. Chronic iridocyclitis and complete occlusion of pupil. Intradermal test with lens protein positive.

Charles S., aged sixty-six, was admitted to the wards of the Massachusetts Charitable Eye and Ear Infirmary May 19, 1921, complaining of failing vision in both eyes of ten years' duration. His history was otherwise negative, except that he had been subject to attacks of asthma, which usually came on in the fall of the year, when hay-fever was most prevalent. He had never had any pain or inflammation in his eyes. The examination was negative at this time, except for double immature nuclear cataracts. V. O. U. = counts fingers at two feet.

May 20: Right eye. Intracapsular extraction attempted, but capsule ruptured. The lens was then extracted by the ordinary method.

May 23: Incision healed. Very little reaction.

May 27: Eye whitening. Good anterior chamber. Bandage off.

May 30: Eye looks whiter each day.

June 4: Patient discharged. V. with sph. + 10 \odot + 1 cyl. axis 15° = 20/30.

June 23: Eye white. Small piece of floating cortical.

July 14: V. O. D. sph. + 10 = 20/70. Thin membrane. Some vitreous opacities. Chronic cyclitis (?).

October 6: Patient states that for the past seven weeks he has had severe inflammation of right eye with slight pain. Treated by local doctor. Pupil closed and drawn up. Tension subnormal. Light projection very poor. Slight pericorneal congestion.

December 2: Eye quiet. Intradermal test with pig lens protein positive.

CASE 12.—Perforating wound of cornea. Traumatic cataract partly

removed by linear extraction. Iridocyclitis. Lens matter removed by irrigation. Rapid recovery. Intradermal tests with lens protein positive.

Louis S., aged thirty-eight, admitted to the hospital March 18, 1921. Eight weeks before was struck in the right eye by a carpet tack. Ten days later was operated upon for cataract. The eye became progressively more and more congested but not painful, and the patient was advised by two ophthalmologists to have it removed owing to danger of sympathetic ophthalmia.

Examination: Moderate pericorneal congestion. The cornea shows a small healed perforating wound near the center and a healed linear cataract incision above. No anterior synechia or evidence of injury to the iris. On the posterior surface of the cornea there are numerous large white precipitates, and there is a small hypopyon (lens matter?). The pupil is contracted and partly obstructed by opaque capsule. No tenderness on pressure. Tension 21 mm. (Souter). Vision = counts fingers at one foot. Left eye normal.

Owing to the absence of any involvement of the left eye in spite of the marked descemetitis in the right, and to the lack of any injury to the uveal tract, sympathogenic uveitis was excluded by Dr. Verhoeff, and a diagnosis made of iridocyclitis due to lens matter.

March 19, 1921: Operation by Dr. Verhoeff. A small keratome incision was made above, and a large amount of lens matter, together with the hypopyon and precipitates on the cornea, removed by irrigation. A large piece of the anterior capsule was removed by forceps. Two hours later the anterior chamber was reformed, the cornea was free from precipitates, and the pupil was clear.

March 22: Some fibrin in pupillary area. No descemetitis.

March 25: Fibrin absorbed. Eye healing well.

April 7: The eye shows evidence of atropin irritation. Hyosein in $\frac{1}{3}$ per cent. solution substituted for atropin.

The eye then made an uneventful recovery, and on May 2 had vision of 20/40 with cataract glass. On July 2 a discission of the lens capsule was performed without reaction following, and on July 13 vision of 20/30 was obtained in spite of the corneal scar.

December 7, 1921: Intradermal test with pig lens protein positive.

Attempting to ascertain the percentage of individuals in general who are hypersensitive to lens protein, we have made dermal or intradermal tests in 50 cases of various eye conditions. These cases were unselected so far as concerned the existence or non-existence of inflammatory reaction dependent upon injury to the lens.

The ages were: Under twenty years, 9 cases; twenty to forty years, 9 cases; forty to sixty years, 8 cases; over sixty years, 24 cases. There were 26 males and 24 females. There were 30 cases in which

there was perforation or rupture of the lens capsule. Four were cases of perforating injury of cornea and lens with retained foreign body which was removed through the anterior chamber, and 26 were cataract cases. Of the cataract cases, four were cases of congenital cataract, in which the eyes were operated upon by discissions; 22 were cases of senile cataract. In 5 of the latter cases cataract extraction by the capsulotomy method had previously been performed, recently followed by discissions, while in 13, extraction by the capsulotomy method was subsequent to the tests. In the remaining cataract cases the tests were made a few days after the cataract extractions.

The tests were definitely positive in only 4 of the 50 cases: one was a case of interstitial keratitis in a female aged thirteen years; one a case of mature senile cataract in a female aged sixty-four, and two were cases of immature cataract operated upon by the intracapsular method, in which the ages were forty-six and sixty-four years. Intraocular inflammation occurred in the two cataract cases, Cases 8 and 10, described above, in which lens matter was left in the eye at operation, and not in the other cataract case in which the lens was removed with its capsule intact.

The important question whether patients who fail to show intraocular reaction following injury to the lens are insensitive to lens protein is answered in the affirmative by the foregoing series of cases, since in the 30 cases of perforation or rupture of the lens capsule inflammatory reaction occurred only in the two cases in which there was a positive skin reaction to lens protein.

To determine whether or not the histologic changes following rupture of the lens capsule in sensitized individuals are such as to indicate that they are due solely to lens matter, we have studied these changes in the five positive cases described above, in which pathologic examinations were made, and compared them with the changes found in sections of all eyes in the pathologic collection of this laboratory which showed injury to the lens. We find that the injured lens in individuals hypersensitive to lens protein attracts chiefly two types of cells, namely, pus-cells and endothelial phagocytes. The pus-cells infiltrated the lens substance, and when lens matter is present in the anterior chamber, collect here also. These cells after a time undergo necrosis, especially when present in abundance. Eosinophiles are generally inconspicuous but are sometimes abundant. The endothelial phagocytes likewise invade the lens

substance and may be seen in the act of phagocytizing it. Sometimes they form thick syncytical masses bordering upon the lens substance, and often form giant cells of the Langhans type. Endothelial phagocytes are also sometimes abundant in the anterior chamber, and here are often loaded with pus-cells and chromatin fragments.

The iris is infiltrated with lymphocytes and plasma cells, the latter greatly predominating, while attached to its surface there are large and small lymphocytes and pus-cells. Similar cells also are collected upon the posterior surface of the cornea where they tend to occur in clumps. The collection of cells on the back of the cornea may be so massive that it causes proliferation of the endothelium and extensive formation of fibrous tissue here (Case 3). Where the process is especially active, there may be a moderate amount of fibrin and blood in the anterior chamber (Case 3).

From the pupillary margin of the iris, fibroblasts and blood capillaries extend over and invade the injured lens and close it off so that ultimately further egress of lens material to the anterior chamber is cut off by fibrous membrane. There is more or less proliferation also of the capsular epithelium, which after a time leads to the formation of anterior capsular cataract. The capsular epithelium in addition seems to give rise to a few free ameboid cells which phagocytize the lens material, but these cells are so similar to the endothelial phagocytes that it is difficult to distinguish them from the latter.

In the cases in which adhesion of the iris to the lens prevented lens material from reaching the vitreous chamber the posterior part of the eye shows little reaction. When there is access of lens matter to the vitreous, the ciliary body shows more or less reaction. The interstitial infiltration, however, is relatively slight, and the infiltrating cells consist almost exclusively of plasma cells. From the epithelial surface there is an exudation of plasma cells and lymphocytes. The ciliary epithelium shows proliferative changes and may give off many free epithelial cells into the vitreous; in Cases 2 and 4 dense cyclitic membranes have been formed. The choroid shows no involvement except anteriorly where the infiltration of the ciliary body may continue into it for a short distance. The retina shows perivascular infiltration with lymphocytes and more or less edema around the disc and in the macular region. In Case 4 there are nodular collections of lymphocytes, plasma cells, and large mononuclear cells on the inner surface of the retina and, in places, localized foci of neuroglia proliferation beneath the surface. The optic disc

shows perivascular infiltration with lymphoid cells, slight edema, and in Cases 1, 2, and 4, neuroglia proliferation along its surface.

In the pathologic collection of the Infirmary, covering a period of twenty-one years, we are able to find sections from 25 cases of injury to the lens in addition to those already described, which show changes which we regard as characteristic of phacoanaphylactic endophthalmitis. We also find sections from ten cases in which we are unable to determine whether or not the inflammatory reaction was due to anaphylaxis. In each of these cases the lens was infiltrated solely with endothelial phagocytes, and in one there was a tubercle on the anterior surface of the iris due to lens matter which had been surrounded by endothelial leukocytes and giant cells. Whether or not in these cases the inflammation was anaphylactic in nature it is difficult to say, but the clinical fact that injury to the lens is often followed by no inflammatory reaction suggests that in some of these cases the reaction may have been the result of low-grade sensitiveness to lens protein. As a matter of fact, three of the patients returned later and each gave a doubtful intradermal reaction with lens protein. Moreover, in the same pathologic collection we are able to find sections from five cases in which injury to the lens had caused no reaction. That so few specimens of this kind can be found is obviously explained by the fact that only under exceptional circumstances are such eyes removed.

ANIMAL EXPERIMENTS

In these experiments our purpose was to ascertain the effects produced by discissions of the lens capsule in normal animals and in animals sensitized to lens protein. Guinea-pigs and rabbits were employed. Ether narcosis was employed in all experiments except three, in which cocain was instilled before operation. All enucleated eyes were fixed in Zenker's fluid and embedded in celloidin. Sections were stained in hematoxylin and eosin, and by Verhoeff's modified Gram stain for bacteria. No bacteria were found except in the right eye of Exp. 1, which was infected with streptococci. In connection with the Gram stain it may be well to state here that the cataractous lens after fixation often in places appears to have undergone transformation into fine granules, some of which are basophilic and stain deeply by the Gram method. Such granules sometimes occur in pus-cells, possibly due to precipitation of lens material within the latter, and often closely resemble large cocci.

In our preliminary experiments $\frac{3}{8}$ gm. of pig lens was injected

subcutaneously into each of four guinea-pigs. All of them died within three weeks, showing that too large a dose had been used. We then reduced the dose to 1/24 gm. per 100 gm. of body weight and had no further lethal effects. Intradermal skin tests were made at the end of four weeks to ascertain whether or not the animals were hypersensitive, and all of the injected animals gave positive tests. One of the control guinea-pigs (Exp. 6) tested in the same way gave a negative reaction. Seventeen days later a discission was performed upon his right lens. This gave rise to so much reaction that we suspected that he had been sensitized by the subcutaneous test previously made. On making another intradermal test we found that this was actually the case, so that we desisted from making these tests in our control experiments. Following are the protocols of the experiments:

EXPERIMENT 1 (Fig. 3).—August 1, 1921: Guinea-pig weighing 515 grams. 5/24 gram of pig lens protein injected subcutaneously.

August 27: Intradermal tests with pig and ox lens markedly positive.

August 30: O. D. Discission.

September 1: Lens cloudy. Slight injection, no marked reaction. Discission opening closing.

September 13: O. D. Discission opening completely closed. Eye quiet.

September 14: O. D. Second discission. Following this operation the eye became infected with streptococci so that further notes upon it are omitted.

September 21: Discission O. S.

September 23: Lens cloudy. Slight pericorneal congestion. Enucleation O. S.

Microscopic Examination.—O. S.: The cornea is edematous and considerably infiltrated with pus-cells. There are also a few pus-cells attached to the endothelium. The filtration angle is free, but contains many pus-cells and endothelial phagocytes filled with pus-cells. These cells are also abundant in the meshwork of the ciliary body. The iris is practically normal, but shows a large posterior synechia on one side. The lens capsule is intact and appears normal except for a gap of $\frac{3}{4}$ mm. wide in the center. Through this gap the lens matter has projected and formed a nodule about 1 mm. in diameter. In the center of the nodule the lens matter is comparatively normal, but becomes more and more cataractous toward the surface, being here broken up into balls and liquefied. The surface of the nodule is covered with a delicate but thick layer of fibrin which completely fills the pupillary area. The lens matter has become invaded with pus-cells which form a definite boundary to the nodule. Numerous pus-cells have also invaded deeply into the nodule, many of them being seen even in the center. At the periphery are also a number of

cells evidently derived from the lens epithelium. Pus-cells also extend through the gap in the capsule for a distance of $\frac{1}{3}$ mm. into the main body of the lens. The latter shows cataractous changes at the periphery, extending backward almost as far as the posterior pole. The capsular epithelium shows marked

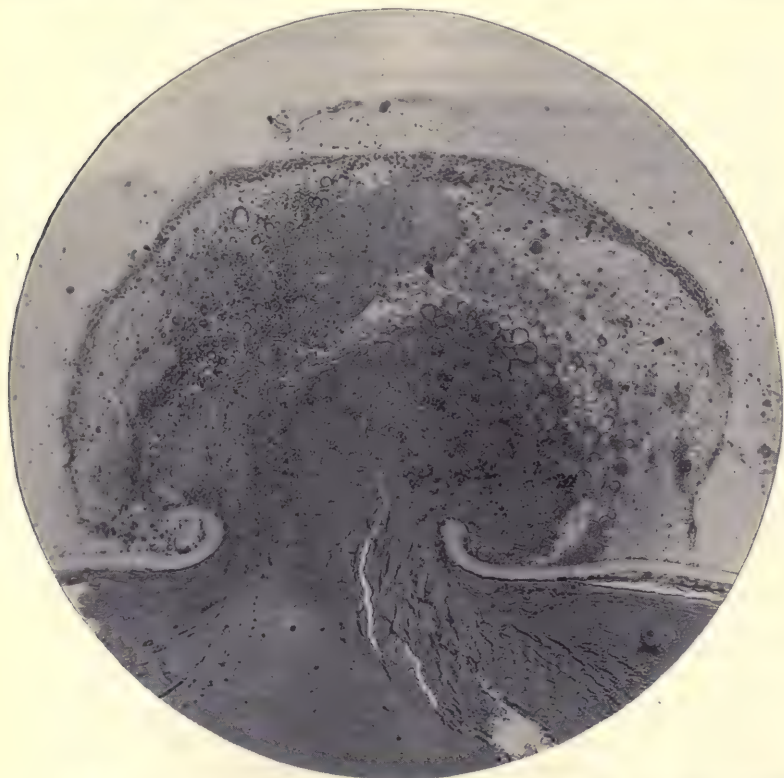


Fig. 3, Exp. 1.—Showing lens changes forty-eight hours after discission of left lens of sensitized guinea-pig. The lens substance has protruded through the gap in the capsule in the form of a nodule. The latter is coated with fibrin and pus-cells, and is deeply invaded by pus-cells, some of which have also extended through the gap in the capsule into the main lens substance. (Photo $\times 75$.)

proliferation, many of the cells being in mitosis, and in places has already trebled in thickness.

EXPERIMENT 2 (Fig. 4).—August 1, 1921. Guinea-pig weighing 470 grams. 5/24 gram of pig lens protein injected subcutaneously.

August 27: Intradermal tests with ox and pig lens protein both markedly positive.

August 30: Discission O. D.

August 31: Lens cloudy. No reaction.
September 5: Eye quiet. Discission opening practically closed.
September 14: Discission opening closed. Second discission O. D.
September 17: No reaction. Discission opening practically closed. Lens slightly cloudy. Third discission O. D.

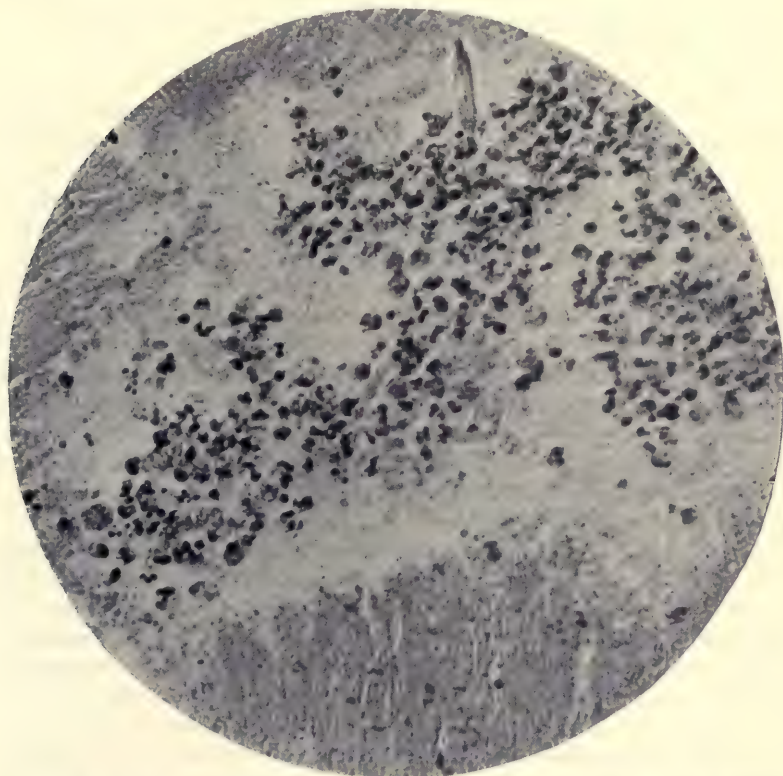


Fig. 4, Exp. 2.—Showing deeply seated purulent infiltrate in lens twenty-seven days after discission of right lens of sensitized guinea-pig. The pus-cells are mostly necrotic. (Photo $\times 400$.)

September 20: Edema of the lids. Circumcorneal congestion. Iris congested. Lens cloudy.

September 22: Pericorneal congestion marked. Vessels have invaded the cornea from above for a distance of 2 mm. Iris congested, markings are indistinct. Vessels are invading the anterior surface of the cloudy lens.

September 24: Cornea becoming vascular all around. Pericorneal congestion not so marked. Iris and lens about the same.

September 26: Eye whiter. Corneal vessels disappearing, also vessels of lens are not so marked. Discission opening practically closed. Iris not so congested. Discission O. S. Enuclation O. D.

October 1: O. S.: Discission opening closed. Lens slightly cloudy. No reaction.

October 5: Second discission O. S.

October 11: Intradermal test with pig lens protein negative. O. S.: Lens nearly clear. No reaction.

Microscopic Examination.—O. D.: The cornea is edematous and thickened to twice its normal thickness. The corneal corpuscles are increased in number throughout and the stroma is moderately infiltrated, chiefly with pus-cells. This infiltration increases as the sclerocorneal margin is approached. Vessels have invaded the whole cornea except a small area 3 mm. in diameter in the center. The endothelium is intact except for a few small areas near the wounds of the discissions. Attached to the lower two-thirds of the endothelium are numerous pus-cells and endothelial phagocytes; many of the latter are in clumps and engorged with pigment and chromatin fragments. The anterior chamber is filled with serum. The filtration angle is open. The meshwork at the angle is infiltrated with pus-cells and endothelial phagocytes many of which are engorged with chromatin fragments and pus-cells. The iris is adherent near the periphery to one of the discission wounds in the cornea, and is adherent on one side to the lens by a large posterior synechia. It is markedly congested and is slightly infiltrated with pus-cells and a few plasma cells. There is a pupillary membrane which is continuous with the pupillary margin of the iris all around and extends over and into the anterior boundary layer. In the center it forms a conical mass which is connected with one of the discission wounds anteriorly. Three openings can be found in the lens corresponding to the three discissions. Through these openings connective tissue extends between the capsule and the lens substance on one side as far back as the equator. Immediately beneath this connective tissue the lens substance shows a large area of liquefaction within which there are large numbers of endothelial phagocytes and pus-cells. Many of the latter are necrotic. Behind this area the lens substance shows deep clefts containing liquefied lens matter, hyaline balls, and many pus-cells which are here especially necrotic. Pus-cells can also be found in small numbers within the solid lens substance throughout its entire thickness. Even when occurring singly these cells lie in small vacuoles evidently due to liquefaction of the lens substance around them. The anterior capsule is about one and three-quarters its normal thickness. Near the discission openings the capsular epithelium is not recognizable, but a short distance from them it appears always at least two cells in thickness, and sometimes in the form of a capsular cataract. At the equator on one side, where the capsule is greatly wrinkled, the capsular epithelium is proliferated into a thick mass in which numerous balls of lens matter have been incor-

porated. Near the posterior pole the capsule shows a gap 1 mm. wide. Here the lens matter is cataractous and markedly infiltrated with endothelial phagocytes and pus-cells.

The vitreous is infiltrated with serum, which is more concentrated posteriorly. In the vitreous, approaching the gap in the posterior capsule, there are many pus-cells and a few lymphocytes.

The ciliary body is infiltrated with plasma cells and lymphocytes throughout, and in places shows nodular collections of these cells.

The surface epithelium shows proliferative changes and is giving off numbers of round cells.

The optic disc is congested and greatly swollen, and is giving off small and large lymphocytes into the vitreous.

The choroid and retina are normal, with the exception that the retina apparently shows edema of the ganglion-cell layer.

EXPERIMENT 3.—August 4, 1921. Guinea-pig weighing 355 grams. 4/24 grams of pig lens protein injected subcutaneously.

August 27: Intradermal tests with pig and ox lens protein positive.

September 19: Discission O. D.

September 21: Lens markedly cloudy. Slight pericorneal congestion.

September 26: O. D. Discission opening closed. Eye quiet. Second discission.

October 1: O. D. Discission opening closed.

October 5: Third discission O. D.

October 8: Pericorneal congestion. Iris congested. Posterior synechia. Ball of cortical matter in pupil. Lens becoming vascular.

October 11: Intradermal skin test with pig lens protein mildly positive.

October 15: O. D. Fourth discission. O. S., first discission.

October 20: O. D. Pericorneal congestion. Slight vascularization of cornea above. Iris congested. A few vessels invading the lens. O. S., no reaction.

October 24: O. D. Descemetitis. Vascularization of the cornea practically cleared. There is an artery that has invaded the lower part of the lens. Iris congested. O. S. no reaction.

October 25: Enucleation O. D.

Microscopic Examination.—Right eye: There is a slight infiltration of the tissues of the limbus with lymphoid and plasma cells. Vessels have invaded the periphery of the cornea for a distance of about $1\frac{1}{2}$ mm. but otherwise the corneal stroma is practically normal. There are a few mononuclear cells attached to the endothelium of the cornea. The filtration angle is free in most of the sections, but in some of them it is occluded by the root of the iris. The uveal meshes are slightly infiltrated with lymphoid cells. The iris stroma, in general, shows very little cellular infiltration, but in the posterior third, in places, there is a considerable number of plasma cells.

A rather delicate pupillary membrane which is continuous with the iris on one side, fills the pupil, and covers the gap in the anterior capsule. This gap is only about 0.15 mm. wide. The anterior capsule is swollen and greatly wrinkled. Tissue from the pupillary membrane accompanied by blood-vessels extends through the gap and mingles with tissue arising from the capsular epithelium. Just beneath this membrane the lens shows a large area of liquefaction containing numerous hyaline balls. This area is markedly infiltrated with endothelial phagocytes, many of which are collected upon and phagocytizing the solid portion of the lens substance bordering upon it. From this liquefied area there extend numerous clefts deeply into the lens substance, some of them reaching almost to the posterior capsule. These clefts are filled with liquefied lens matter markedly infiltrated with pus-cells, but contain few, if any, endothelial phagocytes. Many of the pus-cells are necrotic. Pus-cells are also found in the comparatively normal lens substance, generally lying in small vacuoles which they have evidently produced. The capsular epithelium has extended around almost the entire circumference of the lens. The posterior capsule shows a small gap which has been closed by proliferation of the capsular epithelium. The underlying lens substance is here liquefied and infiltrated with endothelial phagocytes, many of which contain pigment.

The ciliary body is considerably infiltrated, chiefly with plasma cells. From its surface there is a slight exudation of lymphocytes.

The optic disc is greatly swollen and congested. It is markedly infiltrated with lymphoid and plasma cells, the latter predominating. From the porus there is taking place an exudation of plasma cells into the vitreous. An exudation of these cells is also taking place from the retina in the vicinity of the disc. The retina is edematous, the edema being most marked near the disc. The choroid is practically normal, except immediately around the disc, where, on one side, it is markedly infiltrated with plasma cells for about $1\frac{1}{2}$ mm. The pigment epithelium at this margin of the disc shows proliferative changes.

EXPERIMENT 4.—August 4, 1921. Guinea-pig weighing 400 grams. 4/24 gram of pig lens protein injected subcutaneously.

August 27: Intradermal skin tests with pig and ox lens protein both positive.

September 19: Discission O. D.

September 21: Lens markedly cloudy.

September 26: O. D. Discission opening closed. Second discission.

October 1: O. D. Discission opening closed. No inflammatory reaction.

October 5: O. D. Third discission.

October 8: Lens cloudy, but no marked reaction.

October 11: Intradermal skin test with pig lens protein negative. Discission O. S.

October 12: 4/24 gram of pig lens protein injected subcutaneously. No immediate symptoms of anaphylaxis.

October 15: No reaction from the injection of the lens protein. Both eyes free from congestion.

EXPERIMENT 5 (Figs. 5 and 6).—August 4, 1921. Guinea-pig weighing 407 grams. 4/24 gram of pig lens protein injected subcutaneously.

August 27: Intradermal tests with pig and ox lens protein both positive.

September 19: Discission O. D.

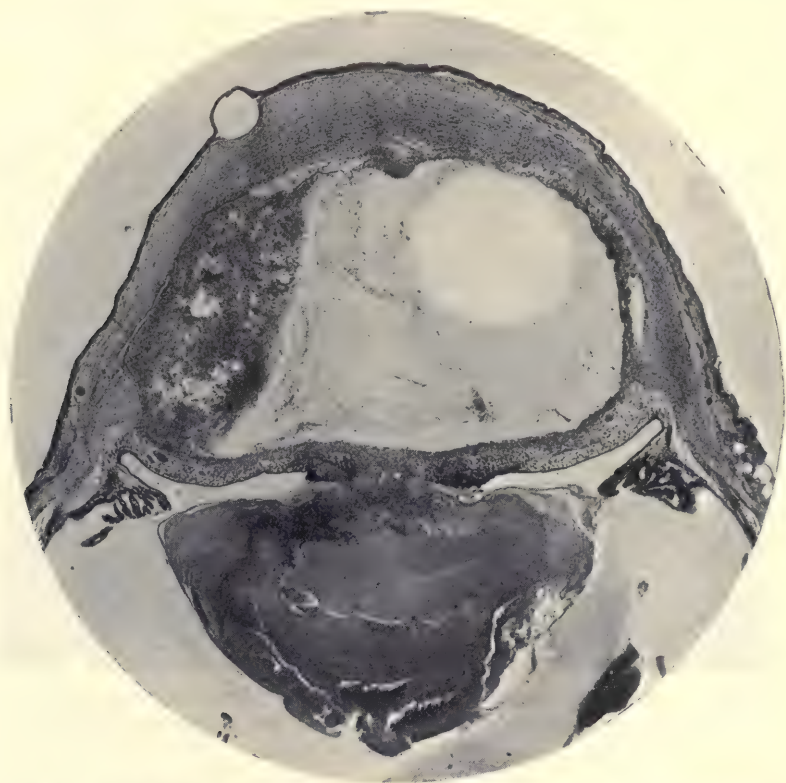


Fig. 5, Exp. 5.—Right eye of guinea-pig, showing marked phacoanaphylactic endophthalmitis eighteen days after discission of lens. Note intense involvement of cornea. (Photo $\times 15$.)

September 21: O. D. Marked pericorneal congestion. Iris congested. Posterior synechia. Lens becoming vascular.

September 22: Eye quieting.

September 24: About the same, except that the cornea is vascularized above for a distance of 1 mm.

September 26: Vascularization of lens and cornea. Otherwise eye is quiet.

September 29: Eye practically white. Vessels of the cornea and lens disappearing.

September 30: O. D. Becoming worse. Pericorneal congestion. Iris congested. Pupil contracted. Vessels invading the cornea all around. Vessels invading the lens.

October 1: O. D. The cornea is completely vascularized, except for an area of about 2 mm. in the center. Edema of the lids. Slight hypopyon.

October 3: Circumcorneal congestion marked. Cornea slightly more vascular. Marked hypopyon. Iris congested. Cornea is bulging. Deep anterior chamber.



Fig. 6, Exp. 5.—Right eye. Showing tissue from pupillary membrane extending into lens, and marked infiltration of lens substance with pus-cells. (Photo $\times 50$.)

October 5: O. D. Cornea presents about the same condition. Hypopyon not so marked.

October 7: Whole cornea is vascular and bulging. Hypopyon about the same. Enucleation O. D. Discission O. S.

October 11: Intradermal test with pig lens protein doubtful. No inflammatory reaction O. S. Lens cloudy.

Microscopic Examination.—O. D.: The cornea is markedly swollen, being quadrupled in thickness in the center. The epithelium is edematous, the basal cells being markedly vacuolated. At one place there is an intra-epithelial cyst, 0.45 mm. in diameter, due to the splitting of the two layers. The stroma

is vascularized to an extreme degree throughout, so that it resembles granulation tissue. There is marked proliferation of the corneal corpuscles. Anteriorly it shows remarkably slight infiltration with pus-cells, but just anterior to Descemet's membrane the infiltration with pus-cells is very marked. Here too the new vessels are especially large and the tissue especially edematous. In two places Descemet's membrane has been destroyed, apparently by the pus-cells within the cornea, and the gap filled by actively proliferating epithelioid cells. There are numerous hemorrhagic extravasations in the stroma, the largest one being just in front of the posterior surface. The endothelium is intact, but it is thickened by proliferation. It is covered by a layer of pus and red blood-cells which is thin above and gradually becomes thicker as the lower angle is approached. At the lower angle it assumes the character of a hypopyon 3 mm. high consisting chiefly of pus-cells. The hypopyon also contains a considerable number of mononuclear cells, endothelial phagocytes, and blood-corpuscles. The filtration angle is completely closed off by a wide peripheral synechia. The pupillary area is filled with dense vascular membrane, rich in fibroblasts, which is continuous with a similar richly vascularized membrane that replaces the anterior surface of the iris everywhere. The iris stroma is moderately infiltrated with plasma and lymphoid cells in the posterior part; as the anterior surface is approached these cells are markedly increased in number. Incorporated within the pupillary membrane are several large masses of cortical substance. These are surrounded by endothelial phagocytes and invaded to some extent by pus-cells. The anterior capsule of the lens shows a gap 1 mm. wide which is filled in by the pupillary membrane. The latter, however, does not extend deeply into the lens. At each margin of the gap the capsular epithelium has proliferated and formed a large mass of tissue which fuses with the pupillary membrane. Immediately behind the tissue filling the gap in the capsule the lens substance is liquefied and is being invaded by pus-cells. The solid lens matter bordering this liquefied area behind is coated with many endothelial phagocytes, and is markedly infiltrated with pus-cells which in places are collected in large masses. On one side the infiltration extends almost to the equator of the lens, through more than half its thickness. The capsular epithelium has not extended posteriorly, and is normal except at the site of the dissection.

The ciliary body is congested, edematous, and infiltrated chiefly with plasma cells. The anterior portion contains a considerable number of endothelial phagocytes many of which are pigmented. The plasma cell infiltration is largely nodular in character and extends as far back as the ora serrata. The ciliary processes are giving off a moderate number of large and small lymphocytes and a few pus-cells. Very few cells are being given off by the pars plana. The optic disc shows edema and congestion, but no significant cellular infiltration. The choroid and retina are practically normal.

EXPERIMENT 6.—August 27, 1921. Guinea-pig weighing 310 grams. Intradermal tests with pig and ox lens protein negative.

August 30: Discission O. D.

August 31: O. D. Lens cloudy. No inflammatory reaction.

September 1: O. D. No reaction.

September 13: Discission opening closed.

September 14: Second discission O. D.

September 16: Marked reaction. Lids slightly swollen. Lens cloudy. Pericorneal congestion. Iris congested.

September 18: Eye clearing up, but lens vascular. Slight vascularization of cornea above.

September 21: Posterior synechia. Anterior surface of lens vascular. Skin test with pig lens protein mildly positive.

September 26: Discission opening not visible. Pupil contracted.

October 5: Vessels in the lens disappearing. Eye white and quiet.

October 11: Iris slightly discolored and congested. Slight pericorneal congestion.

October 15: Slight vascularization of the periphery of the cornea. Vessels in anterior surface of the lens more marked.

November 25: Eye white and quiet. Enucleation O. D.

Microscopic Examination.—O. D.: The cornea appears normal except that it is vascularized with a few small vessels which have invaded the whole cornea except an area 2 mm. in diameter in the center. The root of the iris closes off the filtration angle all around. At the angle the uveal meshes are slightly infiltrated with plasma and lymphoid cells.

The pupillary margin is connected with one of the discission wounds by a long thin strand, rich in connective-tissue cells. The pupillary area is filled by a delicate vascular membrane which is continuous with the pupillary margin of the iris and adherent to the markedly wrinkled lens capsule. This membrane in its anterior half is densely infiltrated with plasma cells. The iris is moderately congested and greatly infiltrated with plasma cells. The lens capsule shows a gap $\frac{1}{3}$ mm. in diameter near the periphery of the anterior surface. This gap is covered by the iris. The epithelium has disappeared from immediately beneath the anterior lens capsule, except for slight stretches here and there. Beneath the capsule there is a wide space filled with delicate avascular tissue which is slightly infiltrated with plasma cells and pervaded by strands of cells derived, evidently, from the capsular epithelium. Bordering this space posteriorly, except for an area in the center, and closely applied to the lens substance, is a layer of epithelium similar to that of a normal lens capsule. At the periphery on one side this layer is continuous with the normal capsular epithelium which is here intact. On the other side the lens capsule is separated still further from the lens substance and is adherent in places to the ciliary processes. The posterior surface of the lens is denuded of capsule

and coated, except for a small area at the posterior pole, by a layer of epithelium continuous with that coating the anterior surface. This, in turn, is coated by a very delicate but thick layer of tissue similar to that beneath the anterior capsule, which, like the latter, is pervaded by epithelial strands. In some sections this tissue continues over the intact capsule at the periphery and becomes adherent to the ciliary processes. It also becomes continuous with the tissue beneath the anterior capsule. In places a few blood-vessels may be seen within it, indicating that it really represents a cyclitic membrane pervaded by epithelial strands derived from the ciliary epithelium. The lens substance is reduced to less than one-half its normal thickness, evidently by absorption. Its central portion is comparatively normal, but the cortex laterally is everywhere cataractous, and is largely converted into bladder cells. The central portion of the anterior surface is coated by proliferated capsular epithelium beneath which is an almost continuous layer of endothelial phagocytes some of which have formed giant cells. The comparatively normal lens substance is infiltrated to a slight extent with pus-cells, many of which are necrotic, while the cortex in places contains numbers of endothelial phagocytes. At the posterior pole the capsular epithelium has formed a tissue similar to that of a capsular cataract, through which endothelial phagocytes are entering the lens.

The ciliary body is congested and shows considerable infiltration with plasma cells which have a tendency to appear in nodules some of which are as far back as the ora serrata. The surface of the processes is giving off a slight exudate containing lymphoid and large mononuclear cells. The cyclitic membrane described above is no longer in active formation.

The optic disc is greatly swollen, and shows moderate infiltration with lymphoid cells and considerable neuroglia proliferation. From its surface there is some exudation of lymphoid and plasma cells. The retina shows slight edema and slight exudation of plasma and lymphoid cells from its surface for a considerable distance from the disc. At the margin of the disc the choroid is considerably infiltrated with plasma cells, scattered foci of which also can be seen as far forward as the equator.

EXPERIMENT 7.—November 25, 1921. Guinea-pig weighing 370 grams. 4/24 gram fresh pig lens protein injected subcutaneously.

December 15: Discission O. D.

December 16: O. D.: Slight pericorneal congestion. Pupil well dilated. Lens matter in pupil.

December 18: O. D.: Slight pericorneal congestion. Pupil not completely dilated. Iris congested. Lens cloudy at point of discission and on posterior surface.

December 20: O. D.: Pericorneal congestion. Pupil contracted. Photophobia. O. S. is dilated to maximum (as a result of transference of atropin from O. D.).

December 21: O. D.: Pericorneal congestion quite marked. Pupil contracted to $1\frac{1}{2}$ mm. Iris congested. O. S. widely dilated. Discission O. S.

December 22: O. D.: Pericorneal congestion. Iris congested. Pupil still contracted. O. S.: Chemosis and congestion of conjunctiva. Pupil completely dilated. Lens cataractous, and a mass of cortical matter projects through the opening in the capsule.

December 23: O. D.: No change. O. S.: Chemosis of conjunctiva marked. Ciliary congestion. Pupil beginning to contract.

December 24: O. D.: Pericorneal congestion not so marked. Pupil contracted. Lens cataractous. O. S.: Chemosis of conjunctiva about the same. Pericorneal congestion marked. Iris congested. Pupil more contracted. Photophobia.

December 25: O. D.: Pericorneal congestion not so marked. Iris and pupil about the same. O. S.: About the same, except that the pupil is more contracted. Enucleation O. U.

Microscopic Examination.—O. D.: The cornea is considerably edematous and infiltrated with pus-cells and shows beginning vascularization. Its posterior surface is coated with lymphocytes. The anterior chamber is filled with serum. There is an anterior peripheral synechia on one side. The iris is adherent at the pupillary margin on one side to the discission wound in the lens, and is congested, but it is very slightly infiltrated.

The discission wound passes entirely through the lens. The gap in the anterior capsule is solidly closed by a mass of cells derived from the capsular epithelium, and the latter is markedly proliferated for a considerable distance on each side of the gap. The gap in the posterior capsule is 4 mm. wide and through it the lens bulges into the vitreous.

The lens is highly cataractous along the path of the discission wound, the cataractous changes being more marked as the gap in the posterior capsule is approached. The lens substance is greatly infiltrated with pus-cells, many of which are necrotic, the infiltration is most marked posteriorly. Pus-cells are also collected in abundance on the posterior surface of the cataractous lens, and it is evidently from here that the infiltration of the lens is chiefly taking place.

The vitreous humor is rich in serum and is greatly infiltrated with pus-cells which become more and more numerous as the gap in the posterior capsule is approached.

From the surface of the ciliary body there is taking place an exudation of pus-cells, lymphocytes, and endothelial phagocytes. The retina and choroid seem practically normal. The optic disc is congested and edematous and is giving rise to a slight exudation of pus-cells and lymphocytes.

O. S.: The cornea is slightly vascularized at the periphery, but is almost free from infiltration. There are a few lymphocytes on the posterior surface. The anterior chamber is filled with serum. The filtration angle is free. The

iris is only slightly infiltrated but shows numerous lymphocytes and a few endothelial phagocytes on its surface.

The discission wound in the lens is very small and does not reach the posterior capsule. The gap in the anterior capsule is only 0.2 mm. wide and is solidly closed by a mass of cells derived from the capsular epithelium to which the iris is adherent. The lens substance along the path of the discission wound is cataractous and greatly infiltrated with pus-cells which are undergoing necrosis.

The ciliary body, especially the orbiculus, is giving off a moderate exudate of small and large lymphocytes. The vitreous is infiltrated with serum and contains large clumps of necrotic large mononuclear cells.

The retina shows slight edema. The choroid is normal. The optic disc is slightly swollen.

EXPERIMENT 8 (Control).—October 10, 1921. Guinea-pig weighing 410 grams. Discission O. S.

October 12: Lens swollen and cloudy. No inflammatory reaction. Enucleation O. S.

Microscopic Examination.—O. S.: The cornea shows a very slight infiltration with pus-cells. Attached to the posterior surface there is an occasional lymphocyte. The filtration angle is open. The iris, ciliary body, retina, and optic disc are normal. The pupil is widely dilated. There is a gap $2\frac{1}{4}$ mm. in the anterior capsule of the lens through which lens substance protrudes forming a nodule $\frac{1}{2}$ mm. high. Along the surface the nodule is slightly cataractous. It is coated with a layer of delicate fibrin which contains an occasional lymphocyte. The capsular epithelium at the margin of the gap is undergoing proliferation and beginning to extend over the surface of the protruding nodule. The cataractous lens matter at the periphery of the nodule contains very few cells. The cells have round or oval nuclei and their cell bodies are swollen. They certainly are not pus-cells and possibly have migrated from the capsular epithelium. The solid lens substance is absolutely free from cellular infiltration.

EXPERIMENT 9 (Control, Figs. 7 and 8).—September 21, 1921. Guinea-pig weighing 440 grams. Discission O. D.

September 24: Eye white and quiet. Lens cloudy.

September 26: Discission opening in capsule closed. Second discission O. D.

September 29: Eye white. Lens cloudy.

October 1: Discission opening closed. Lens cataractous at posterior pole only.

October 5: Third discission O. D.

October 8: O. D.: Eye quiet. No congestion. Lens cloudy.

October 10: O. D.: Eye quiet. Discission opening closed. Discission O. S.

October 12: Both eyes quiet. Lenses cloudy.

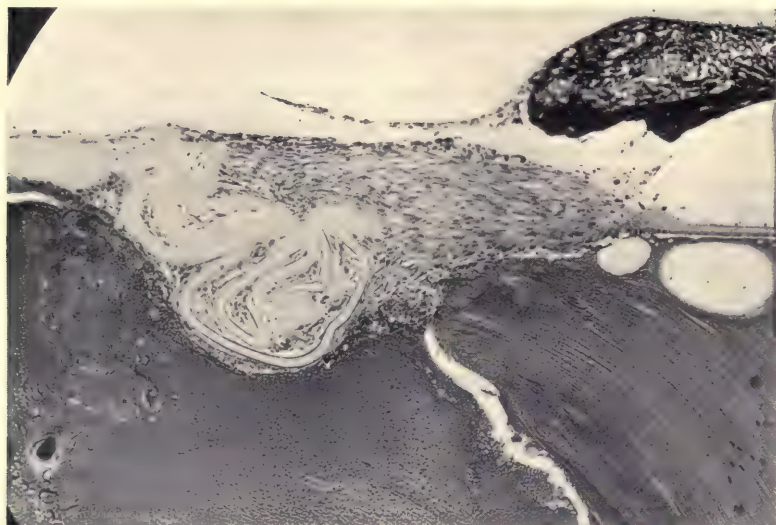


Fig. 7, Exp. 9.—(Control.) Showing discission wound nine days after third discission in right eye of non-sensitized guinea-pig. The wound is closed by proliferation of the capsular epithelium and the iris on one side only is lightly adherent to the mass of tissue thus formed. The lens substance is free from infiltration. (Photo $\times 75$.)

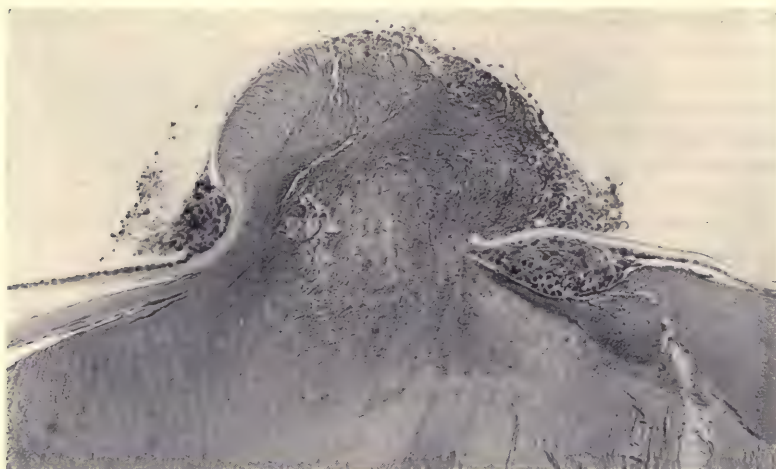


Fig. 8, Exp. 9.—(Control.) Showing lens changes four days after discission of left lens of non-sensitized guinea-pig. The protruding nodule is coated with a delicate layer of fibrin and a few cells derived from the capsular epithelium, but is free from infiltration with pus-cells. (Photo $\times 75$.)

October 14: No change. Enucleation O. U.

Microscopic Examination.—O. D.: The cornea is absolutely normal. The anterior chamber contains a trace of serum. Filtration angle open. The iris, ciliary body, and retina are free from infiltration. The optic disc is possibly slightly swollen and shows a small interstitial hemorrhage, but is free from cellular infiltration. An occasional lymphocyte may be seen upon the surface of the ciliary body. The capsule of the lens shows a gap about $\frac{1}{4}$ mm. wide. This is completely closed by a thick mass of tissue rich in cells, due to proliferation of the capsular epithelium. The pupillary margin is slightly adherent to this mass. The underlying lens substance is moderately cataractous for a microscopic distance, and infiltrated to a slight extent with free cells from the capsular epithelium which are undergoing necrosis, but is free from pus-cells. The posterior capsule is ruptured and the lens substance posteriorly has swollen to such an extent that it has enlarged the gap in the capsule to a width of 4 mm. The peripheral portion of the lens thus exposed is highly cataractous, being liquefied and broken up into hyaline balls. The lens substance bordering on the cataractous portion contains an abnormal number of nuclei, apparently produced by proliferation of the nuclei of the lens fibers, but is free from cellular infiltration, as is also the cataractous portion of the posterior cortex. The vitreous is infiltrated with serum, but is free from cellular infiltration.

O. S.: Except for the lens, the eye is normal. The lens capsule shows a gap 0.7 mm. wide through which the lens substance protrudes in the form of a nodule about 0.5 mm. in height. Along its surface the nodule is broken up into hyaline balls which are covered by a thin layer of fibrin. The capsular epithelium has proliferated and extended as an irregular layer of cells for a short distance over the surface of the nodule. The anterior surface of the fibrin shows an occasional thin, elongated cell with an ovoid nucleus. The substance of the nodule is infiltrated very slightly with free cells apparently derived from the capsular epithelium, many of which show fragmentation of their nuclei. The main lens substance is apparently normal.

EXPERIMENT 10 (Control).—August 27, 1921. Guinea-pig weighing 380 grams. Intradermal tests with pig and ox lens protein negative.

September 19: Discission O. D. The animal made a sudden movement and the knife went entirely through the lens, causing a large hemorrhage in the vitreous.

September 21: Lens cloudy. No inflammatory reaction.

September 24: Discission opening closed. No reaction.

September 26: Second discission O. D.

September 27: No reaction O. D. Skin test with pig lens protein doubtful.

October 1: Discission opening closed. Lens cataractous in posterior portion only.

October 5: Third discission.

October 8: Eye quiet. Lens cloudy. No reaction.

December 5: At no time has the animal had any intraocular inflammation. O. D.: Enucleation.

Microscopic Examination.—The cornea and iris are normal. There is no peripheral anterior synechia, posterior synechia, or pupillary membrane. The discission wound in the anterior capsule is closed by proliferation of the capsular epithelium. The underlying lens substance appears normal for a considerable distance, then cataractous changes may be seen along the path of the discission wound. Here also, deep in the lens, there are a few small collections of cells which are so necrotic that their nature cannot be determined. Just behind the equator on one side the posterior capsule shows a wide gap and the lens substance is coated by a thick layer of spindle-cells which is continuous with the capsular epithelium at the equator. Beneath this the lens substance shows numerous bladder cells. Upon the posterior surface of the lens there is a moderate amount of blood which has attracted a few endothelial phagocytes and lymphocytes. The ciliary body is free from infiltration, but on the side nearest the gap in the posterior lens capsule it is coated with a small amount of blood. From its surface there is a slight exudation of endothelial phagocytes and lymphocytes which evidently have been attracted by the blood.

The choroid and retina are normal. The physiologic cup of the optic disc is filled with lymphocytes and endothelial phagocytes. Some of the latter contain blood pigment, indicating that the presence of these cells is the result of the hemorrhage in the vitreous.

EXPERIMENT 11.—December 10, 1921. Guinea-pig: Discission O. D. The cornea about the discission wound immediately became cloudy from infiltration with aqueous humor.

December 12: No pericorneal congestion. No inflammatory reaction. Cornea cloudy about the wound. Pupil well dilated. Lens cloudy.

December 13: O. D. Eye quiet and white. Cornea still cloudy. Pupil completely dilated. Discission O. S.

December 14: O. D. Eye quiet. Pupil completely dilated. Cornea the same. Lens slightly cloudy. O. S. No congestion. Pupil dilated to maximum. Slight cloudiness of the lens with a mass of cortical substance protruding into the anterior chamber.

December 15: O. D. Cloudiness of cornea practically gone. Eye quiet. O. S. Eye quiet.

December 19: Both eyes white and quiet. Discission opening appears closed. Lens matter in anterior chamber O. S. completely absorbed. Both pupils dilated to maximum.

December 21: Both eyes quiet. Pupils still dilated.

December 23: No change. Enucleation O. U.

Microscopic Examination.—O. D.: The discission wound is closed by a mass

of cells continuous with and evidently derived from the capsular epithelium. The lens substance is free from infiltration with pus-cells or endothelial phagocytes. The condition is essentially the same as in Exp. 9 (Fig. 5), but the iris is not adherent to the lens. The lens substance shows only slight cataractous changes, repair evidently having taken place. The eye is otherwise normal.

O. S.: The gap in the capsule is 0.75 mm. wide and is filled in by a mass of epithelial cells through the center of which a strand of lens substance still projects. The iris is adherent to this mass. The underlying lens substance is slightly cataractous and slightly infiltrated with free epithelial cells, most of which are necrotic. There is no infiltration with pus-cells. The eye is otherwise normal.

EXPERIMENT 12.—November 25, 1921.: 4/24 gram pig lens protein injected subcutaneously.

December 15: 4.20 P. M. 4/24 gram pig lens protein injected subcutaneously.

4.22 P. M.: Animal is restless.

4.24 P. M.: Animal quiet and appears very sick.

4.30 P. M.: Animal cuddles up to other animals as if cold. The hair begins to stand erect.

4.35 P. M.: Has difficulty in standing and incontinence of urine.

5.20 P. M.: Still has incontinence of urine and difficulty in standing. Hair still stands erect.

7.30 P. M.: Animal walks about, but hair is now erect on the face so that eyes appear sunken. Marked lacrimation.

10.30 P. M.: Animal able to walk about and hair not so erect.

December 16, 1921, 7.30 A. M.: Animal appears normal except that the hair is slightly erect.

EXPERIMENTS ON RABBITS

Discissions of the lenses were made in the eight eyes of four animals. In two of the animals the discissions were made twenty-one days after subcutaneous injection of 1/24 gm. of fresh pig lens protein per 100 gm. of body weight. The eye of one of the control animals was infected, and is therefore excluded from consideration. Clinically, the intraocular reactions were definitely greater in the treated animals than in the controls, but in the latter were greater than in control guinea-pigs. Histologically, the reactions were also more marked in the injected animals than in the controls, but did not show the characteristic features seen in hypersensitive guinea-pigs and human individuals. For this reason we omit the detailed protocol

of these experiments. Possibly sensitization by repeated small doses of lens protein might give more characteristic results.

The foregoing clinical observations, tests, and microscopic examinations prove conclusively that certain individuals are hypersensitive to lens protein, and that when in such individuals rupture of the lens capsule takes place through injury or operation or spontaneously, a characteristic inflammatory reaction results. This reaction may properly be termed endophthalmitis phacoanaphylactica.¹ Whether or not hypersensitiveness to lens protein is congenital or acquired, and if the latter, how it is acquired, we have at present insufficient data to determine. While experimentally it is possible to sensitize an animal to its own lens protein, the evidence is against the possibility that injury to the lens of one eye may sensitize an individual so that a local anaphylactic reaction will take place if the other lens is injured. The clinical fact that in cases of congenital cataract discission of the cataract in the second eye ordinarily causes no greater reaction than that following discission of the cataract in the first eye seems to be sufficiently conclusive on this point. Possibly in these cases absorption of lens matter slowly and continuously over a long period of time, if it has any effect, may cause immunity rather than hypersensitiveness.

Our experiments upon animals seem to show that a guinea-pig may be sensitized to lens protein so that a free discission of its lens capsule will be followed by a severe and characteristic intraocular reaction. That the dosage of lens protein employed was sufficient to sensitize the animals is shown by the skin tests and also by Exp. 12 in which a second subcutaneous injection made 20 days after the first caused marked general symptoms of anaphylaxis. Histologically the ocular reaction differs from that in human individuals only in the extent to which the cornea is affected, the latter, in the case of the guinea-pigs, showing marked haziness and vascularization. In Experiments 2 and 5 the cornea became almost completely vascularized. It is to be noted, however, that in one of our clinical cases, Case 1, the cornea became almost completely vascularized. In our control experiments the intraocular reaction following discission of the lens was not only slight, but differed in character from that seen in a sensitized animal. As already pointed out, one animal (Exp. 6) intended as a control, became sensitized by an intradermal test and gave a phacoanaphy-

¹ Endophthalmitis phacoallergica may be a better term, but probably it would not be generally understood.

lactic reaction following dissection of the lens. In Experiments 2, 3 and 5 no reaction followed dissection of the lens of the second eye made 19 to 26 days after dissection of the lens of the first eye. It is probable either that these animals had become immunized as a result of the first dissections and intradermal tests, or that sufficient time had elapsed for the hypersensitiveness to have disappeared, for in Exp. 2, a negative, in Exp. 3, a mildly positive, and in Exp. 5, a doubtful intradermal reaction was subsequently obtained. In Exp. 4 no inflammatory reaction followed dissections of the lenses of an animal supposed to be hypersensitive, but in this experiment an intradermal test made later was negative, and the animal failed to show any general symptoms of anaphylaxis after a subcutaneous injection of lens protein.

As Longcope⁸ has recently pointed out, in human individuals the state of hypersensitiveness to foreign proteins shows certain differences from that produced experimentally in animals, and he raises the question whether it is based upon the same mechanism. In this connection he calls attention to the familiar occurrence of hypersensitiveness to certain proteins in human individuals, which suggests that inheritance is an important factor, and to the rarity with which specific antibodies are found in the serum of these individuals. Whether or not, however, there is an important difference between the mechanisms of natural and artificially produced anaphylaxis, the clinical effects seem to be essentially the same, and our experiments on guinea-pigs, as just noted, seem to show that in animals sensitized to lens protein the intraocular reaction produced by injury to the lens is microscopically identical with that in human individuals. It has been frequently noted that immune and anaphylactic reactions of rabbits are markedly different from those of guinea-pigs, and Zinsser⁹ has recently pointed out the close similarity of such reactions in guinea-pigs and human individuals. The former fact no doubt explains our failure to produce typical phacoanaphylactic endophthalmitis in rabbits, and the latter fact is confirmed and amplified by the positive results of our experiments on guinea-pigs.

The clinical picture of phacoanaphylactic endophthalmitis is fairly characteristic, especially in marked cases. In the beginning there is simply iritis with posterior synechia. From the pupillary margin a vascularized membrane extends over the injured lens. At the same time the lens matter becomes infiltrated until it presents an opaque, finely granular appearance. At this stage precipitates may be found

on the posterior surface of the cornea. These are apt to be unusually large and white in appearance. Sometimes a small hypopyon is formed. If the process continues, the cornea becomes cloudy and progressively vascularized. The intraocular tension is apt to be increased. Pain and congestion of the eye vary with the activity of the process and are sometimes relatively slight.

To make the diagnosis certain, dermal and intradermal tests with lens protein should, of course, be employed in all cases.

Our observations concerning the histology of phacoanaphylactic endophthalmitis are sufficiently summarized above, but we may point out here that the invasion of the lens substance by pus-cells seems to be especially characteristic, particularly as regards the relatively normal portion. In other words, in hypersensitive individuals exposed lens matter is pyogenic. We should, in fact, hesitate to make a diagnosis of phacoanaphylactic inflammation histologically, in the absence of any purulent infiltration of the lens. The severity of the intraocular reaction depends largely on the extent of the gap in the lens capsule and upon the amount of lens substance that is extruded into the anterior chamber or vitreous. When the gap in the capsule is so situated that access of lens matter to the vitreous is early cut off by adhesion of the iris to the lens, the reaction is confined chiefly to the anterior part of the eye, notably the iris. When the lens matter accumulates in the vitreous chamber, cyclitis results, and may lead to the formation of dense cyclitic membranes. In this event also the retina and optic disc may be markedly affected. When the defect in the lens capsule is covered over by organized tissue derived from the iris, egress of lens material is prevented and the inflammatory reaction then takes on a chronic and probably intermittent character, and may persist for a year or more.

The microscopic examination in Case 2 shows that there had been an old rupture (traumatic?) of the lens capsule and old severe iridocyclitis. As a result of the latter, the lens had been walled off and invaded by dense connective tissue. The lens cortex had become completely cataractous and in many places calcified but the nucleus had caused a marked inflammatory reaction, as shown by the fact that it was surrounded by endothelial phagocytes and giant cells and deeply invaded by an abundance of pus-cells. Whether or not the original iridocyclitis represented a reaction to lens matter it is, of course, impossible to say, but there can be no doubt that the recent reaction was of this nature. This case seems to show that reaction to lens matter may recur at long intervals.

From the foregoing observations it will be seen that phacoanaphylactic endophthalmitis, both clinically and histologically, produces a picture similar to, if not identical with, that of the so-called serous traumatic iridocyclitis. We are, however, not yet prepared to say that the latter condition is always phacoanaphylactic in origin.

After cataract extraction in hypersensitive individuals the severity of the reaction depends upon the amount of lens matter left in the eye and upon the extent to which it is protected by the collapsed capsule. In some cases the reaction may subside while a large amount of lens substance still remains closed off within the capsule. A dissection of the lens capsule will then lead to a renewed inflammatory reaction.

It is probable that the severity of phacoanaphylactic endophthalmitis also may partly depend upon the degree of sensitization to lens protein. Our clinical data, however, are insufficient to establish this as a fact, but it is indicated by the results of the histologic examinations, as noted above.

Judging by our skin tests, individuals hypersensitive to lens protein are more sensitive to normal lens matter (immature cataract) than they are to the cortex of a fully mature cataract, and our histologic findings seem to indicate that in hypersensitive individuals normal lens matter is more pyogenic than is cataractous lens matter.

Our tests made upon human subjects afford interesting confirmation of the facts, previously demonstrated in other ways by animal experimentation, that lens protein is not species specific, and that an individual may be hypersensitive to the protein of his own lens. For we found that patients hypersensitive to pig lens were also hypersensitive to ox lens and human lens, and that the one individual hypersensitive to pig lens who was tested with protein from her own immature cataract, gave a positive reaction to the latter.

The sera of human individuals hypersensitive to foreign proteins seldom give positive complement fixation or precipitin tests. Miss Helen May, of the Massachusetts General Hospital, has kindly made these tests in two of our cases in which markedly positive intradermal reactions had previously been obtained. Pig lens was used as the antigen. Both tests were negative in all dilutions. It would seem, therefore, that such tests are of no practical value in determining hypersensitiveness to lens protein. Possibly, however, they may prove of value in determining the success or failure of attempts to immunize hypersensitive patients.

In cases of hypermature cataract the fluid cortex is said to be especially irritating to the eye. We have had opportunity to examine

microscopically two eyes with Morgagnian cataracts in which there had been rupture of the lens capsule without history of injury. The changes in each eye differed only in degree. In neither eye was the nucleus infiltrated with pus-cells. The cellular exudate which came chiefly from the iris, consisted exclusively of endothelial cells, except in the more marked case in which there was an insignificant collection of pus-cells at one place on the iris. The endothelial phagocytes were collected in abundance on the anterior surface of the iris and on the posterior surface of the cornea. The iris was free from infiltration with lymphocytes and plasma cells, and in the more marked case had undergone almost complete necrosis. In this case there was also hypopyon of 1.5 mm. composed entirely of endothelial phagocytes, mixed with a slight amount of blood. In the other case also there was a slight amount of blood in the anterior chamber. These findings are markedly different from those in our cases known to be hypersensitive to lens protein, as well as from those in our series of untested cases in which the cataract was not hypermature, and strongly indicate that the irritation to the eye produced by Morgagnian fluid is not a reaction of anaphylaxis but is analogous to that produced by any necrotic tissue.¹ If the patient should happen to be sensitive to lens protein there would, of course, be superimposed a phacoanaphylactic reaction.

The treatment of phacoanaphylactic endophthalmitis should consist in the use of atropin to dilate the pupil and of hot fomentations to control the pain. If the patient proves to be sensitive to atropin, hyoscin may be employed instead.² If only a small amount of lens matter remains in the eye, or the latter is well protected by the capsule, no other treatment may be needed. If the condition does not subside within a reasonable time, a linear extraction should be performed and the lens matter thoroughly removed by irrigation (Case 12). This procedure should also be employed in cases in which the intraocular pressure becomes greatly elevated.

In cases which have reached a chronic stage owing to the lens matter having been closed off by fibrous tissue, the process could, perhaps, be checked by desensitizing the patient by means of repeated

¹ Since this was written one of these patients has returned and submitted to an intradermal test with lens protein. The result of the test was negative.

² It may be well to mention here that in at least five of our positive cases the patient became sensitive to atropin. Whether or not this is of any special significance in connection with the fact that atropin is an antianaphylactic, we are at present unable to say.

injections of lens protein. Probably, however, linear extraction would be preferable in these cases also.

In connection with prophylaxis, tests with lens protein preceding operation would seem to be of considerable value, especially in cases of congenital and immature senile cataract. In cases of fully mature senile cataract, or of sclerosed lenses, a positive test would emphasize the importance of removing all cortical matter possible. In cases of congenital cataract it would indicate the employment of free discission followed by early linear extraction, or, possibly, an attempt to desensitize the patient to lens protein before operation.

In cases of immature senile cataracts, a positive test would indicate the advisability of postponing operation, if possible, until the cataracts had fully matured. When this would entail too much loss of time, and the patient was over fifty years of age, an intracapsular operation should be performed upon one eye, and the cataract in the other allowed to become mature.

In the case of a patient under fifty years of age with immature cataract it might be well, owing to the danger of rupture of the capsule, to attempt to produce immunity to lens protein before operation. After such immunity had been established, it might be possible in such a case to employ discission of the cataract followed by simple linear extraction. (Since this was written we have succeeded in desensitizing such a patient, judging by the fact that he now gives a negative skin reaction, whereas he gave an intensely positive test before treatment with lens protein. The details of this case will be reported later.)

SUMMARY AND CONCLUSIONS

In a series of 12 cases of rupture of the lens capsule, traumatic, operative, or spontaneous, in which there occurred intraocular inflammation apparently due to lens matter, a positive skin reaction to lens protein was obtained in every case.

Microscopic examination of five eyes from these cases showed a characteristic histologic picture in each.

In a series of 28 cases of rupture of the lens capsule in which there occurred no intraocular inflammation a negative skin reaction to lens protein was obtained in every case.

In a series of 50 unselected cases of various kinds there was a positive skin reaction to lens protein in four cases.

An individual hypersensitive to lens protein of the ox or pig is also hypersensitive to the protein of his own lens.

Experiments on guinea-pigs seem to show that free discission of the lens capsule of an animal sensitized to lens protein causes a marked intraocular reaction with characteristic histologic changes.

Inflammation resulting from lens matter in the eyes of individuals hypersensitive to lens protein, we have termed phacoanaphylactic endophthalmitis.

Intraocular inflammation resulting from rupture of a Morgagnian cataract is not usually phacoanaphylactic in nature.

All patients with congenital or immature cataracts should be tested with lens protein before operation.

In the case of a patient with immature cataracts in both eyes, and over fifty years of age, who is found hypersensitive to lens protein, the cataract in one eye should be removed in capsule, or operation postponed until it is fully mature. If the patient is under this age, an attempt should be made to desensitize the patient before operation.

The best treatment for severe phacoanaphylactic endophthalmitis is removal of the lens matter by operation.

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DISCUSSION

DR. LUCIEN HOWE (Buffalo, N. Y.): If any of you are interested in the subject I would ask you to look at the rabbits which Prof. Guyer has brought, and I would be glad to have those who pass through Buffalo, especially our foreign friends who go to Niagara Falls, stop to see my little menagerie of twenty or thirty, and learn from them something additional of this ophthalmic aspect of serology.

The practical application of what we have heard seems to be that it is your duty and mine to test every cataract patient in the manner described before operating.

Another point which we too often neglect is great thoroughness in washing out the wound—irrigation—in every case whether there is the least trace of cortical substance visible or not.

PROFESSOR G. F. ROCHAT (Groningen, Holland): I would like to ask first about the dislocation of the lens. Perhaps Dr. Verhoeff might be able to tell us why the dislocated lens has such a deleterious effect on the eye. All of us know that after the lens has been thrown into the vitreous there is some secondary inflammation, and I have never been able, even with a microscopic section, to find a definite reason. It may be that in the solution of the lens material in the vitreous the protein substance of the lens causes inflammation. I have made no special experiments upon that subject. Perhaps Dr. Verhoeff is better informed than I am.

I have noticed in a few of his cases there was trauma—the capsule of the lens was ruptured. Might it be possible that the removal of the lens—these occurred in aged people—gave rise to a certain irritation, and perhaps that would be a better explanation than the anaphylactic explanation Dr. Verhoeff has given us.

DR. JAMES M. PATTON (Omaha, Neb.): Our experience in the use of Dr. Verhoeff's method has been limited to one case, and, although the result was negative, it was scarcely a fair test as the reaction present may have been secondary to previous traumatism. I am reminded of three cases which showed decided postoperative irritation and which might have been foreseen had these tests been used. These cases were unsuitable for ordinary cataract extraction, due to vitreous degeneration, and an attempt to remove the cataracts by discission was followed in each case by marked irritation. Later on in these cases at irregular intervals there were periods of inflammation apparently secondary to throwing out of broken-down lens substance in the aqueous.

It seems to me that Dr. Verhoeff has given us a very valuable preoperative test that will show us, in those cases which are unsuitable for operation, whether we may safely proceed with the discission operation. If this is true, then we can employ it and be able to give the patient an idea as to what the outcome may be.

DR. L. C. ROOD (Boston, Mass.): In consideration of this excellent paper it is striking to note the freedom of the ciliary body from participation in the morbid process excepting where lens matter has access to the vitreous space,

and that when the ciliary body is involved this process is but slightly interstitial, consisting mostly of plasma cell invasion. Also that the slight affection of the choroid, excepting in parts proximal or contiguous to the source of irritation, contrasts strongly with sympathetic ophthalmia, in which condition the choroid is so selectively affected. Also that in these cases of endophthalmitis occurring after extraction, the endophthalmitis occurs after those operations in which masses of cortical matter and capsule remain—a big argument in favor of intracapsular extraction. It is also striking in several cases described in this paper in which an intracapsular operation was successfully done in the second eye, thus removing all cortical matter from the second eye, that the morbid process subsided in both eyes, because the amount of lens substance capable of absorption was lessened and therefore the anaphylactic dosage lessened. It occurs to me that many instances of this type of case (following trauma causing lens injury with consequent affection of the second eye) must have been clinically considered and diagnosed as sympathetic ophthalmia—an entirely different process. Also that cortical matter enclosed in capsule (Case 9) proved an irritant and produced a morbid process even as when free. Case 10 shows strikingly that this irritation is due to the presence of cortical matter, and subsides when the cortex is totally absorbed or removed.

DR. ARNOLD KNAPP (New York City): I have only one question, and that is why is lens matter so well borne by children and young adults?

DR. GEORGE S. DERBY (Boston, Mass.): Dr. Verhoeff supplied me with material to do some of these tests, and although my results have been few in number, they were rather striking. So far as I can understand there is a difficulty in keeping the lens protein material for testing in a sterile condition, and therefore the question arises of interpretation of the test, as to whether it is a positive test from the effect of the lens material or whether it may be a slight infection in doing the skin reaction.

DR. A. N. LEMOINE (Boston, Mass.): We wish to enumerate a few observations made since writing the paper.

In spite of every precaution it has been found that it is extremely difficult to preserve a stock solution of lens protein without it becoming contaminated, consequently in our recent work we have been preparing a fresh solution each time as it was needed. To facilitate this preparation the powder is now collected in the usual way, then put up in amounts of 0.003 gram in a sterile sealed ampule. These ampules can be made by drawing out a three-eighth inch test tube at its center, then adding the weighed quantity of lens powder, and breaking and sealing the tube in the drawn portion. When a solution is desired, the top of the tube is broken, and one c.c. of sterile saline added. This makes a 3.3 per cent. solution of powdered lens, or a 10 per cent. solution when computed in terms of fresh lens protein. This method of preparation does away with the addition of preservatives which precipitate a portion of the protein in the solution and thereby leaves a portion of the protein altered.

Our subsequent observations and experiments have convinced us that there

are variations in degree of hypersensitivity and sensitization. Reactions which were considered doubtful in our former observations are now called mildly positive, since in these cases when the lens capsule is injured and there is free cortical matter, the eye becomes quite irritable and convalescence retarded, even though no active iridocyclitis is present.

To facilitate the interpretation and recording of the findings the following terminology has been adopted for the intradermal test made with a 3.3 per cent. solution of powered lens protein:

- + + + = a reaction with an area of erythema over six or seven cm. in diameter with considerable elevation at the point of injection.
- + + = a reaction with an erythema between three and six cm. in diameter and a moderate elevation at the point of injection.
- + = a reaction with an area of erythema about two to three cm. in diameter with a slight elevation at the point of inoculation.
- ± = a reaction with an area of erythema less than two cm. in diameter with very little elevation at the point of inoculation.
- = when the control and test areas are practically the same.

Skin tests were made on seventy-five unselected cases with much the same results as obtained in our first fifty cases. Following is a summary: Below 30 years of age, 18 cases; 30 to 45 years of age, 13 cases; 45 to 60 years of age, 19 cases; over 60 years of age, 25 cases. There were forty-three males and thirty-two females. Of this number there were seven cases which gave a positive intradermal test to lens protein. Two were males and five were females. The ages were fifty, fifty-five, sixty-eight, seventy, seventy-two and seventy-six. There were also six other cases which gave a mildly positive (±) intradermal test. Four were males and two were females. The ages of the latter cases range from thirty-two to seventy-two years. From these figures as well as those given in the paper, it seems that the hypersensitiveness occurs only in the later decades of life.

Our subsequent animal experimentation seems to verify our previous findings, except that we have not been able to reproduce experiment No. 5.

DR. F. H. VERHOEFF (closing): I wish to emphasize the fact that our conclusions are based solely on our observations in human cases. While our animal experiments seem conclusive also, we find it is impossible to rule out entirely the possibility of slight infection playing some part in the reaction in animals.

In regard to inflammatory reaction, our observation has been that unless the test is definitely positive, injury to the lens will not cause inflammatory reaction of a serious nature.

The lens substance consists of three proteins. We employed the whole lens substance, but it may prove of value to separate these proteins and ascertain whether there are immunologic differences between them.

In answer to Prof. RoCHAT as to dislocation of the lens, we have not had a case of that kind to test. There is a great variability in these cases as to the amount of reaction. Sometimes it is very slight, sometimes a good deal. In the few we have examined microscopically the dislocated lens has caused

pigmentary changes, at the ora serrata below, as in the case of foreign bodies, and I suppose it is the repeated traumatization over a long period of time that causes these changes.

In answer to Dr. Knapp's question about children, the reason they do so well is because they are not hypersensitive. We have not run across a case of congenital cataract that was hypersensitive.

In view of Prof. Guyer's experiments it would be well to make the intradermal tests on the parents of individuals who have congenital anomalies of the eye. But there are a good many questions to be worked out in this connection.

SERUM AND VACCINE TREATMENT FOR THE PREVENTION AND CURE OF CATARACT

A PRELIMINARY REPORT WITH THE RESULTS OBTAINED IN 13 CASES

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Some twenty years ago the author conceived the idea that the cataractous lens in the human eye might be dissolved or absorbed by means of a cytolytic serum; and it is interesting to note here some experiments which were made at the Pediatrics Laboratory, in West 54th Street, New York City, in 1902 and 1903, under the direction of the late Dr. Justin DeLisle. The sera used were: (1) Rabbit's serum immunized against human cataractous lenses. (2) Rabbit's serum immunized against normal guinea-pig lenses.

EXPERIMENT 1.—March 20, 1902. Rabbit No. 1: Three guinea-pig lenses freshly cut out and pulped up finely in 3 c.c. of normal saline solution were injected into a large white rabbit (peritoneal injection); March 31, April 14 and 24, the same amount of lens material was injected; on April 14 and 24 subcutaneously. May 1, bled the rabbit and secured 63 c.c. of clear serum. This serum was hermetically sealed in an ampule.

July 18, 1902: Fresh serum was taken from a rabbit which had not been immunized, 5 c.c. of this fresh serum was added to 5 c.c. of the immunized serum of rabbit No. 1 in a test-tube into which a freshly extracted lens from a guinea-pig was added. The tube was sealed with cotton and placed in an incubator at 37° C. July 20, lens partly absorbed; July 22, lens continues to diminish in size; July 28, about one-third of the lens is digested and the fluid has turned a chocolate color; July 31, lens remains about the same size and the serum a little darker. All but the nucleus of the lens has been absorbed.

EXPERIMENT 2.—Sept. 23, 1902. Rabbit No. 4: A medium-sized female.

A crystalline lens of a guinea-pig was placed under the skin of rabbit No. 4 by dissecting up the skin of the belly, inserting the lens, then sewing up the skin. October 3 a second guinea-pig lens was inserted under the skin of the rabbit, also on October 14. On October 23 and November 5 two crystalline guinea-pig lenses (each date) were introduced, and on November 15 and 29 two guinea-pig lenses (each date) were pulped in normal saline solution and injected under the skin. December 13 the rabbit was bled and about three ounces of blood obtained. December 24, $3\frac{1}{2}$ c.c. of the serum obtained from this blood was injected under the skin of a large pregnant female guinea-pig (No. 1) which had a mature senile gray cataract in each eye. December 25 guinea-pig aborted a litter of pigs; all died. December 29, little or no change in the cataractous lenses of the mother pig. December 31, injected $3\frac{1}{2}$ c.c. more of the sensitized rabbit (No. 4) serum into the guinea-pig (No. 1). January 1, 1903, the cataracts are not so gray, but of a silky luster, as if water was mixed among the fibers; January 6, lenses pellucid and not so gray; January 6 and 8 (each date), 2 c.c. of serum from rabbit No. 4 was injected into guinea-pig No. 1 and the cataractous lens in the guinea-pig No. 1 continued to clear slowly.

In the meantime rabbit No. 4 was again injected with guinea-pig crystalline lenses, subcutaneous injections of the pulped lenses being given on December 24, 1902, January 8, 1903, and the rabbit was bled on January 20, 1903, and allowed to die. January 25, 1903, injected $3\frac{1}{2}$ c.c. of this fresh serum from rabbit No. 4 subcutaneously into guinea-pig No. 1. January 30, the cataractous lens in the left eye of guinea-pig No. 1 continues to clear and the right also, but not so rapidly. On February 3 and 11 (each date) $2\frac{1}{2}$ c.c. of serum from rabbit No. 4 was injected subcutaneously into guinea-pig No. 1. On March 6, 1903, no further absorption of the cataractous lenses in the guinea-pig taking place, two minims of the sensitized serum from rabbit No. 4 were injected into the anterior chamber of the left eye of guinea-pig No. 1. March 7 the lens was swollen and the capsule ruptured: slight redness of eyeball. The entire cortex of this cataractous lens was absorbed but not the nucleus.

EXPERIMENT 3.—Rabbit No. 5: Male, large size. October 14, 1902, this rabbit was immunized to human crystalline lens by inserting human cataractous lenses (whole) under the skin and by pulping human cataractous lenses in normal saline solution and injecting same under the skin. October 14 and 23, each date, one human lens was inserted under the skin. November 15 and 29, each date, one human lens pulped and injected subcutaneously. January 29, rabbit was bled and let die. Six drams of serum was secured, which was heated for one hour at $55\frac{1}{2}^{\circ}$ C., then sealed in an ampule. February 17, 1903, an untreated rabbit, No. 6, was bled for fresh serum. In a sterilized test-tube one human cataractous lens was placed, and to this was added 2 c.c. of fresh serum from rabbit No. 6, and to this 1 c.c. sensitized serum from rabbit No. 5. In a second test-tube 1 c.c. of fresh serum from

rabbit No. 6 and 2 c.c. of sensitized serum from rabbit No. 5, to which was added one human cataractous lens. The tubes were sealed with cotton and placed in a sterilizer at 37° C. February 18, tube No. 1: the lens is softened and partly absorbed. Tube No. 2: the lens is more softened and there is more absorption than in the lens of tube No. 1. February 25, tube No. 1 continued absorption of the lens, about one-third being absorbed. March 6, slight further absorption of lens in each tube. On account of evaporation of the fluid, 5 c.c. of serum from rabbit No. 6 (freshly taken) was added. In all the cortex of the lens was absorbed, but not the nucleus.

It will be noted in the above experiments that an attempt was made to dissolve or absorb the guinea-pig cataractous lens, both *in vitro* and *in vivo*, with anti-lens serum, and that in each instance only a partial success was obtained. With the human cataractous lens the attempt to dissolve the lens was made only *in vitro*, as I must admit I did not have the courage to try the experiment at that time on the living subject. The above are a few experiments conducted at that time (1902-3), crude, it must be admitted, and only partially successful.

Because of inadequate laboratory facilities and equipment, and without any encouragement whatsoever to continue these experiments, so very little being known about the subject at the time, reluctantly the work was given up for a short time, as I supposed, though the idea continually lay in the back of my mind. It was not until seventeen or eighteen years later, when the articles of Guyer and Smith on "Studies on Cytolysins"¹ were published, that my enthusiasm was again stimulated into action. These investigators succeeded in liquefying the lenses of the young *in utero* of rabbits and mice by injecting anti-lens serum into the mothers, and, what is more, succeeded in transmitting the eye defects so produced into succeeding generations—the special object, by the way, of their experiments. It is true that they did not succeed, in any instance, in liquefying, absorbing, dissolving, or affecting in any way apparently the lenses of the adult rabbits or mice they experimented upon. However, later on in this paper more will be said on this point.

Perhaps before giving the actual experiments conducted on the human subjects it would be well to consider briefly the agents—cytolytic sera and vaccines—with which the experiments were made. In my earliest experiments—1902-03—of necessity I was compelled to make my own sera; in fact, to make the bleeding tubes, ampules, etc., by which the serum was obtained. In the present experiments

¹ The Journal of Experimental Zoölogy, May, 1918, to August, 1920.

Dr. Russell L. Cecil has assisted me in the preparation of all the sera and vaccines, and I am further indebted to Dr. Cecil for many valuable suggestions during the course of these experiments. The method of preparing the sera and vaccines is given elsewhere in this paper.

CYTOLYSINS

Cytolytic sera are produced by injecting tissue cells (antigen) of one species, *e. g.*, red blood-cells, nerve tissue, crystalline lens, etc., into the blood of another species. A lytic substance (amboceptor or antibody) is thus formed which is called a *hemolysin* when red blood-cells are used, a cytotoxin or cytolsin when other tissue cells are used as the antigen. These lytic substances (cytolysins) thus formed are specific in the main for the antigen (cells) used to produce the cytolsin. Most of the cytolysins are *species* specific; that is, they react only on the species from which the tissue or antigen is obtained. A few of the cytolysins are *organ* specific to a greater or lesser degree, *e. g.*, spermatozoa, the testicle, the ovary, and of the tissues of the eye, the crystalline lens and the uveal pigment. Very recently Hektoen¹ has called attention to the specific precipitin reaction of the lens, and cites Uhlenhuth, who first called attention to the fact that the lens of different species give the same immune reactions, that is, that it is *organ* specific and not *species* specific. Hektoen (*loc. cit.*), after numerous experiments with lens antigens of different animals, arrived at the conclusion that the *organ* specificity of the lens holds good throughout and that the lens does not appear to contain any *species* specific antigens; hence the lens protein may be regarded as chemically distinct and as identical in diverse species. Hektoen also claims that, "So far the lens is the only clean-cut example we have of this organ specificity as contrasted with species specificity in antigen-antibody reactions." Bearing directly on this point Zinsser has this to say: "Recent critical study of these organ-cytotoxins (cytolysins) has revealed, however, that the specificity of a serum produced with the tissues of one organ is not strictly limited to this organ alone, and that the serum may injure other organs as well."

Guyer and Smith (*loc. cit.*) also call attention to the fact that all cytolytic sera so far studied have been found to be more or less hemolytic in action. Like the hemolysins, they are also specific in action on the special tissue used as the antigen in the sensitizing

¹ Hektoen: Jour. Amer. Med. Assoc., July 2, xxi, 32; also Amer. Jour. Oph., December 21, xxi, 909.

process. However, they are not absolutely specific in action, as homologous tissues may be affected to a slight extent, *e. g.*, the vitreous when lens cells are used as antigen. According to Ehrlich and his school, lack of absolute specificity is due to the fact that homologous tissues may have common receptors. It is interesting to note in this connection a statement of Hektoen's: "The aqueous and vitreous humors appear frequently to contain lens substance, because in most instances these fluids in low dilutions or full strength react with lens antiserum, but says nothing about the aqueous. These humors may react with serum antiserum also—not always—and then the law of species specifiveness obtains. It is true, of course, that in some of my specimens lens substance may have become mixed with either humor as a result of the handling incidental to the withdrawal of the humor, but I believe in most instances this did not occur, as the withdrawal was made with special care to avoid undue pressure." Incidentally it may be stated here that the other tissues of the eye of organ specificity is the uveal pigment. As is well known, Elschnig¹ based his theory of sympathetic ophthalmia on the anaphylactic action of this pigment, and later Alan C. Woods,² of Baltimore, has added to the knowledge on this subject by his studies and experiments at Johns Hopkins Hospital, Department of Ophthalmology.

Bearing in mind all the limitations of specificity of action of the different cytolytic sera so far developed, and the dangers that may be incurred by their lytic action on the cells of the homologous tissues other than the tissue cells furnishing the antigen, the experiments on the human subject (related in detail below) were proceeded with on the basis that the main action would be on the tissue cells used as antigen, especially on organ specific antigens.

There is one other danger also which I would point out here, and that is anaphylactic shock, more or less severe in character, which at times follows the intravenous injection of the lens antiserum and lens vaccines. More will be said about this later.

¹ Elschnig, A.: Studien zur sympathischen Ophthalmie: I. Wirkung von Antigen vom Augeninnern aus, *Arch. f. Oph.*, 1910, lxxv, 459; II. Die antigene Wirkung des Augeninnern aus, *ibid.*, 1910, lxxv, 459; III. *ibid.*, 1911, lxxviii, 549; Elschnig, A., and Salus, R.: IV. Wirkung des Augenpigments, *Arch. f. Oph.*, 1911, lxxix, 428.

² Woods, A. C.: The Anaphylactic Basis of Sympathetic Ophthalmia, *Tr. Sect. Oph.*, A. M. A., 1917, 133-161; Woods, A. C.: Ocular Anaphylaxis. V: Experimental Iridocyclitis, *Arch. Oph.* (March), 1918, xlvii, 161. Woods, A. C.: Immune Reactions Following Injuries to the Uveal Tract.

EXPERIMENTS WITH CRYSTALLINE LENS

CHEMICAL COMPOSITION OF THE CRYSTALLINE LENS.—The greater part of the solids of the crystalline lens consists of proteins. The actual percentage of the various solids in beef-lens is as follows:

Protein.....	35.00
Fat and lipin.....	0.75
Salts.....	0.75
Water.....	63.5

The protein constituents have been divided by Mörner into two parts—the albumoids and the crystallins. Albumoid is found in the lens fibers and is insoluble in water and in salt solution. It dissolves readily in dilute acids or alkalis. The crystallins (α - and β -crystallin) are globulins, and are soluble in water and in salt solution.

Jess has shown that in the senile cataract of oxen the crystallins are transformed in part to albumoid. According to Hammarsten, the relation between albumoid and crystallins is changed with increasing age, the albumoid increasing, the crystallins decreasing. In normal lens the reaction of the crystallins to the albumoid changes correspondingly from 82: 18 in youth, to 41: 59 in old age; in senile cataract the relation can be changed to 25: 75. The other constituents of the lens remain unchanged.

PRESENT EXPERIMENTS.—The first experiments were concerned with the effect of intravenous injections of anti-lens serum on patients suffering with cataract. The anti-lens serum was prepared in three different ways:

1. By immunizing rabbits against normal guinea-pig lenses (anti-guinea-pig-lens rabbit serum).
2. By immunizing rabbits against human cataract lenses (anti-human-cataract-lens rabbit serum).
3. By immunizing a sheep against normal beef lenses (anti-beef-lens sheep serum).

In view of the great similarity in chemical composition of cataract lens to normal lens, there seemed to be no reason for confining the study to the effect of anti-cataract-lens serum. The comparative scarcity of cataracts would have made such a study impracticable; hence the use of sera from animals immunized with normal guinea-pig or beef lenses.

Later on the unpleasant effects of the serum led to a study of the effects of lens antigen when injected directly into patients with cataracts. This also appeared more rational on theoretic grounds, as more

lens antibody was probably made available by active immunization than by attempts to confer a passive immunity with serum.

PROTOCOLS

1. PREPARATION OF ANTI-GUINEA-PIG-LENS RABBIT SERUM.—Fresh guinea-pig lenses were removed under sterile precautions, ground into a mortar, and suspended in normal salt solution in the proportion of one lens to each cubic centimeter of salt solution. The suspension was then centrifuged to remove coarse particles, and the supernatant opalescent fluid preserved in vaccine bottles. To insure sterility, a few drops of chloroform were added to each bottle and the preparation stored in the refrigerator.

Two large rabbits were injected intravenously every day for six days with small doses of guinea-pig lens extract as follows:

Rabbit 56: Male, 2511 gm. Received 0.5 c.c. lens extract intravenously on January 10 and 11, then 1 c.c. of lens extract intravenously every day from January 12 to January 15 inclusive.

Rabbit 57: Male, 2825 gm. Received lens extract intravenously every day for six days; dosage same as for rabbit 56.

January 25, ten days after last injection, rabbits 56 and 57 killed by bleeding from carotid arteries.

Serum preserved.

2. PREPARATION OF ANTI-HUMAN-CATARACT-LENS RABBIT SERUM.—Human cataract lenses which had been recently removed at operation were dried over anhydrous calcium chlorid, then ground to a powder in a sterile mortar. The powder was then suspended in normal salt solution in the proportion of one lens to 2 c.c. of saline, the coarser particles removed by centrifuge, and the supernatant fluid decanted into sterile bottles. A few drops of chloroform were added for preservative.

Two large rabbits were injected intraperitoneally once a week for six weeks with 5 c.c. of the lens extract as follows:

Rabbit 52: 1477 gm. Received 5 c.c. of cataract-lens extract intraperitoneally December 14, 1921, and 5 c.c. every week thereafter until January 17, 1922.

Rabbit 53: 1620 gm. Received 5 c.c. of cataract-lens extract intraperitoneally December 14, 1921, and 5 c.c. every week thereafter until January 17, 1922. January 24 both rabbits were bled to death and their serum preserved on ice.

Two other rabbits were injected with cataract-lens extract intravenously. The injections were given daily for seven days. Dosage: 0.5 to 1 c.c. of extract.

Rabbit 58: 1878 gm. Received 0.5 c.c. cataract-lens extract intravenously on February 7 and February 8. February 9–13, the daily dose was increased to 1 c.c. of extract.

Rabbit 59: 1952 gm. Received 0.5 c.c. cataract lens extract intravenously on February 7 and February 8. February 9-13, the daily dose was increased to 1 c.c. of extract.

February 23, rabbits 58 and 59 bled to death and serum preserved on ice.

The following rabbits were also injected with cataract-lens extract intravenously:

Rabbit 60: Male, 2261 gm. Received cataract-lens extract intravenously on seven successive days. Dosage: March 1 and March 2, 0.5 c.c. extract; March 3-8, 1 c.c. extract; March 12, bled to death. Serum preserved.

Rabbit 61: Female, 2647 gm. Received cataract-lens extract intravenously on seven successive days (March 1-8). After an interval of one week the rabbit received another series of five injections on successive days (March 15-19).

Dosage: March 1-2, 0.5 c.c. extract; March 3-8, 1 c.c. extract; March 15-19, 1 c.c. extract; March 26, bled to death. Serum preserved on ice.

Rabbit 62: Male, 2474 gm. Received cataract-lens extract intravenously on six successive days (March 14-19).

Dosage: March 14-15, 0.5 c.c. extract; March 16-19, 1 c.c. extract; March 26, bled, but not killed.

One month later (April 19) a second series of injections was started. This time the injections were given intravenously at four- to six-day intervals.

Dosage: 1 c.c. of extract. Number of injections in second series, 13. (April 19, 26, 30; May 3, 9, 13, 17, 21, 25; June 2, 6, 10, 16.)

June 27, bled to death. Serum preserved.

Rabbit 63: Male, 2052 gm. Received cataract-lens extract intravenously on six successive days (March 14-19).

Dosage: March 14-15, 0.5 c.c. extract; March 16-19, 1 c.c. extract; March 26, bled, but not killed. One month later (April 19) a second series of injections was started. This time the injections were given intravenously at four- or five-day intervals.

Dosage: 1 c.c. of extract. Number of injections in second series, 12. (April 19, 26, 30; May 3, 9, 13, 17, 21, 25; June 2, 6, 10, 16.)

June 27, bled to death. Serum preserved.

Rabbit 64: Female, 2070 gm. Received cataract-lens extract intravenously on four successive days (April 5-8).

Dosage: April 5-6, 0.5 c.c. extract; April 7-8, 1 c.c. extract.

Second series of injections, also intravenous, on four successive days.

Dosage: April 12-15, 1 c.c. extract; April 20, bled, but not killed.

Third series of intravenous injections at four- to six-day intervals.

Dosage: 1 c.c. of extract. Number of injections, 6. (April 26, 30; May 3, 9, 13, 17.) May 20, rabbit bled and serum preserved.

Rabbit 65: Male, 1915 gm. Received cataract-lens extract intravenously on four successive days (April 5-8).

Dosage: April 5-6, 0.5 c.c. extract; April 7-8, 1 c.c. extract.

Second series of injections, also intravenous, on four successive days.

Dosage: April 12-15, 1 c.c. extract; April 20, bled, but not killed.

Third series of intravenous injections, April 26-May 17

Dosage: 1 c.c. of extract. Number of injections, 6. (April 26, 30; May 3, 9, 13, 17.) May 20, rabbit bled and serum preserved.

Rabbit 66: Male, 2308 gm. Received cataract-lens extract on seven successive days. All injections intravenous.

Dosage: May 19-20, 0.5 c.c. extract; May 21-25, 1 c.c. extract.

Second series started June. Three intravenous injections of 1 c.c. each at four-day intervals.

June 20: Rabbit bled and serum preserved on ice.

Rabbit 67: Female, 2147 gm. Received cataract-lens extract intravenously on seven successive days.

Dosage: May 19-20, 0.5 c.c. extract; May 21-25, 1 c.c. extract.

Second series started June 2. Three intravenous injections of 1 c.c. each at four-day intervals. June 20, rabbit bled and serum preserved on ice.

Rabbit 68: Female, 2476 gm. Received cataract-lens extract intravenously on seven successive days.

Dosage: May 19-20, 0.5 c.c. extract; May 21-25, 1 c.c. extract.

Second series started June 2, consisted of three intravenous injections of 1 c.c. each at four-day intervals.

June 20: Rabbit bled and serum preserved on ice.

Rabbit 69: Female, 1931 gm. Received cataract-lens extract intravenously on seven successive days.

Dosage: May 19-20, 0.5 c.c. extract; May 21-25, 1 c.c. extract.

Second series started June 2, consisted of three intravenous injections of 1 c.c. each at four-day intervals.

June 20, rabbit bled and serum preserved on ice.

3. PREPARATION OF ANTI-BEEF-LENS SHEEP SERUM.—Fresh beef lenses were removed under sterile precautions and dried in a vacuum oven at 35° C. The dried lenses were then pulverized in a sterile mortar and the powder preserved in sterile glass bottle.

The lens extract was prepared as follows:

Powdered lens	10.0 gm.
Sodium carbonate	0.5 gm.
Sodium chlorid	0.85 gm.
Distilled sterile water	100.0 c.c.

The solution was centrifuged to remove coarse particles, and the opalescent extract preserved by the addition of a few drops of chloroform.

A sheep was injected intravenously every four to seven days with the beef-

lens extract. From time to time the sheep was bled and the serum preserved for further study and for therapeutic inoculations.

Sheep protocol follows:

Sheep No. 1: Female, full grown, received beef-lens extract intravenously: on September 21, 23, 2 c.c.; September 27, 3 c.c.; and on October 3, 7, and 13, 4 c.c. each day. Followed immediately by symptoms of anaphylaxis. On October 19 received 2 c.c. extract intravenously. No symptoms; October 25, bled from jugular vein. Serum preserved. On October 27, 3 c.c. extract intravenously; November 4, 2 c.c.; November 10, 1 c.c.; November 16, 2 c.c.; November 21, 2 c.c. On December 4, 350 c.c. blood removed from jugular vein. Serum preserved; December 7, 4 c.c. extract *subcutaneously*.

PROTOCOLS OF PRECIPITIN TESTS

1. PRECIPITINS IN RABBIT SERUM FOR CATARACT-LENS PROTEIN.—The serum from rabbit 65 was tested for precipitins against cataract lens after having received 12 intravenous injections of human cataract-lens extract.

The tests were performed as follows:

Various dilutions of lens extract (10% solids) were made with normal salt solution, and this served as the antigen.

The rabbit serum was diluted in the usual way (1:10, 1:100, 1:1000, etc.).

Equal parts of extract and diluted serum (0.5 c.c. each) were mixed and incubated for two hours at 37° C.

The results follow:

Immune rabbit serum—	1:10	1:100	1:1000	1:10,000	1:100,000	Saline
Cataract-lens anti-						
gen..... 1:10	++	++	+	—	—	—
1:100	+	+	—	—	—	—
1:1000	+	—	—	—	—	—

REPORT OF CASES.—All of these patients were inmates of the State Hospital for the Insane, Central Islip, N. Y. All injections of serum and vaccine were administered intravenously, except when otherwise stated.

CASE 1.—Elizabeth S., admitted to the hospital May 21, 1920, aged eighty-three; nativity Germany; white, housework, widowed. Physical symptom complex: general arteriosclerosis, enlargement of the heart, impairment of the hearing; mature senile gray cataract right eye; projection good, pupils active, anterior chamber normal in depth, tension normal each. This patient was the first to receive anti-human-lens rabbit serum. The serum was administered intravenously on the following dates: February 1,

21, 3 c.c., from rabbit No. 52; February 6, 11, 15, 5 c.c., from rabbit No. 52; February 20, 24, 5 c.c., from rabbit No. 53; March 3, 7, 13, 20, 23, 5 c.c., from rabbit No. 59; March 28, 5 c.c., from rabbit No. 60; April 1, 7, 13, 5 c.c., from rabbit No. 61; April 18, 5 c.c., from rabbit No. 61.

The patient vomited five minutes after the injection. April 22: Examination of the eye shows wedge-shaped sectors of translucency forming in the upper half of the lens, the edge of the wedge being at the center of the lens. April 22: 5 c.c. from rabbit No. 61; April 28, 5 c.c. (mixed) from rabbits Nos. 62 and 65. Patient vomited five minutes following injection, but showed no other ill effects. Patient put to bed on account of general weakness. May 6, 5 c.c. (mixed) from rabbits Nos. 62 and 65. May 9: Patient had an attack of syncope due to general arteriosclerosis. She became weak on her way to the dining-room and had to be returned to bed. May 11: 4 c.c. (mixed) from rabbits 62 and 65, no reaction followed. May 16: 5 c.c. (mixed) from rabbits 62 and 65; patient showed slight reaction at the time but did not vomit as on the two former occasions when she had reactions. Later in the day the patient died from a stroke of apoplexy. At the time of the death of this patient the upper half of the lens was semitranslucent (pellucid in appearance), the outer cortical layers apparently had become liquefied. Unfortunately, no postmortem was allowed, and the eye could not be obtained for examination under section. Because of the advanced age, the pronounced arteriosclerosis, and enlargement of the heart, this patient was an unfavorable subject for the treatment, and we have not given the injections to any but the physically sound (except moderately arteriosclerotic subjects) since. We do not attribute the fatal attack to the treatment, as on May 9, three days after any injections had been given, the patient had an attack of syncope, and all but passed away.

It would have been highly interesting and most instructive to have made sections of the crystalline lens and found out what physical changes had taken place in its structure.

CASE 2.—Susan A., admitted July 2, 1921, aged about sixty years, American, housewife, good physical condition. Mature senile gray cataract, left eye; projection good, anterior chamber normal in depth left, slightly shallow in right; tension normal each, pupils active. Serum was administered intravenously on the following dates: July 10, 15, 20, 3 c.c. from rabbit No. 66. On July 20, within two minutes after the injection, the patient complained of nausea, excruciating intermittent pain low down in the lumbar region. The head and neck became congested and, a little later, a marked lividity followed. She experienced no difficulty in breathing and the pulse was regular and of good quality. After seven minutes, all symptoms of distress subsided and patient was allowed to sit up. Because of this marked reaction to the serum-lens vaccines were substituted in place of serum injections (beef-lens 10% in solution with sodium carbonate 0.5%, sodium chlorid 0.9%, and chloroform q. s. to preserve). July 26, 31, August 2, 1 c.c. beef-lens vaccine.

August 5, 10, 15, 20, 25, 31, 2 c.c. beef-lens vaccine. September 10, 2 c.c. beef-lens vaccine. Examination of the left eye shows wedges (prism-shaped sectors) of translucency in nasal and temporal halves, horizontal meridian, of the left lens, the bases of the wedges are about 1 mm. in width and come to a point at the center of the lens, cortical layers alone being affected. Incipient cataract in right eye unchanged by the treatment. September 15, 1 c.c. beef-lens vaccine was given intradermally for sensitization, a pronounced redness and irritation of the skin was produced at the site of the injection. September 20, 21, 2 c.c. beef-lens vaccine; September 25, 30, October 5, 3 c.c.; October 10, 15, $3\frac{1}{2}$ c.c.; October 20, 30, 3 c.c. The translucent sectors in the lens continue to increase in size, and a fresh sector has appeared in the lower half. November 4, 9, $\frac{1}{2}$ c.c. anti-beef-lens sheep serum. Following the second injection there was a marked general reaction—

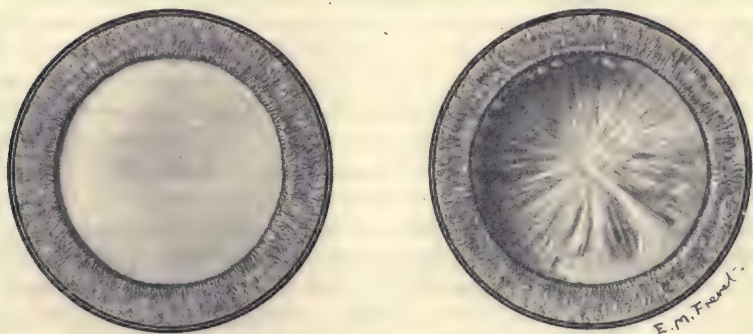


Fig. 1.—Case 2. Mrs. A. L. E., two stages.

flushing of the skin, pain in the back, some weakness, moderate elevation of the temperature, and six hours later fall in the blood pressure. Before treatment 118–218, six hours later dropped to 100–190. November 14, treatment omitted. Because of the marked reaction the patient was again placed on the beef-lens vaccine. November 16, $\frac{1}{2}$ c.c.; November 19, $\frac{1}{2}$ c.c.; November 21, following last injection, patient complained of intermittent pain in the back and a desire to urinate frequently for first few hours after the injections; temperature rose to 100° and pulse to 90. Today gave 1 c.c. beef-lens vaccine, no pain or reaction of any kind followed. November 29, 2 c.c. beef-lens vaccine; blood pressure before injection 100–200, after, 110–210. One minim of the vaccine was given intradermally, and marked local reaction followed. November 30, temperature 100, no other symptoms. December 4, 1 c.c. beef-lens vaccine; within three minutes excruciating pain in the back, involuntary movements of bladder and bowels, and slight diffuse

redness of the skin. One hour later temperature, 100; pulse, 80; respiration, 26. December 9, because of the severe reaction, it was decided to stop intravenous injections and to give the vaccine subcutaneously, beginning with 1 c.c. and increasing the dose $\frac{1}{2}$ c.c. at a time. December 14, 1 c.c. beef-lens vaccine subcutaneously; December 19, $1\frac{1}{2}$ c.c.; December 24, 2 c.c. Immediately before injection B. P. 180-90, pulse 90. Six hours later B. P., 210-90; pulse, 90. Differential blood count: No. cells, 270; poly., 56.7; lym., 38.5; l. mon., 1.9; eos., 2.2; bas., .4; hem., 65%.

No reactions so far from the subcutaneous injections. The lower half of the lens in the left eye has a bluish, translucent, wedge-shaped sector, having coalesced except one fibrous sector, which extends directly downward to VI on the clock dial. With the ophthalmoscope there is a very faint reflex through this portion of the lens. The right lens apparently has not been affected one way or the other, and vision remains 20/40. (See Fig. 1.)

CASE 3.—Catherine C., admitted August 3, 1921, aged about seventy-seven years; some arterial thickening, impaired hearing, cataracts in each eye, mature right, incipient left, of the dark sclerosing type; anterior chambers, pupils, and tension normal; good light projection. August 8, 1 c.c. serum intravenously from rabbit 69; August 15, 2 c.c. serum intravenously from rabbit 69. Complained of feeling slightly dizzy, but showed no objective symptoms. Statement unreliable owing to mental condition. August 20, 2 c.c. serum from rabbit No. 69. A slight amount of this injection was given subcutaneously before the needle entered the vein. On August 22 there was marked redness and swelling of the forearm and hand; August 24, still complains of soreness and stiffness of hand and arm, but redness and swelling subsiding. General condition good. August 25, gave 1 c.c. intravenously serum from rabbit No. 69. Two minutes later the patient complained of excruciating pain across the hips, and about four hours after the treatment she fainted. During the attack she vomited and the circulation was poor. Two hours later still extremely irregular and slow pulse, 54 per minute, and complained of weakness. All treatment was suspended for about one month. September 20, 25, 1 c.c. 10% beef-lens vaccine; September 30, $\frac{1}{2}$ c.c. 10%; October 5, $\frac{1}{2}$ c.c. 10%; October 10, 15, $1\frac{1}{2}$ c.c. 10%. Patient complained of nausea and gastro-intestinal disturbance after the last injection. October 20, 30, 1 c.c. beef-lens vaccine. Examination of the eyes shows no distinct changes in the lens opacities. November 4, $\frac{1}{4}$ c.c. anti-beef-lens sheep serum. Marked reaction within five minutes, tachycardia, retching, complaint of burning sensation throughout the body, anxiety, dyspnea, surface of the body flushed at first, then bluish in color, especially about the nose and mouth, recurrent vomiting began after the injection and lasted for 12 hours. All serum injections were discontinued. November 16, returned to the 10% beef-lens vaccine— $\frac{1}{2}$ c.c. being given. November 19, $\frac{1}{2}$ c.c.; November 24, 1 c.c.; November 29, 2 c.c. Blood pressure before injection 70-130, after injection 80-120. Two drops of the vaccine given intradermally; no local

reaction noted. November 30, temperature, 98; pulse, 76; headache, sickness at the stomach, vertigo, but no vomiting. Because of continued reactions intravenous injections were discontinued and the vaccines were given subcutaneously. December 4, 9, $\frac{1}{2}$ c.c. beef-lens vaccine; December 14, 1 c.c.; December 19, $1\frac{1}{2}$ c.c.; December 20 patient complained of nausea and vomited. Examination of the eye reveals practically no changes in the cataracts. December 24, 2 c.c. beef-lens vaccine. Immediately before injection, blood pressure, 140-85; pulse, 84; six hours after blood pressure 124-80; pulse, 84. Differential blood count: No. cells, 250; poly., 64.4; lym., 28; l. mon., 4.8; eos., 2.4; bas., .4; hem., 80%.

CASE 4.—Mary S., admitted August 24, 1904, now aged seventy-one years. General condition only fairly good; right femoral hernia, varicose veins in lower extremities, impairment in hearing and sight. Mature senile gray cataract right eye, immature nuclear cataract left, also choroidal changes noted in the fundus. Anterior chamber normal depth right, slightly shallow left, pupils and tension normal, light projection good. August 5, 1 c.c. serum from rabbit No. 66; August 10, 15, 20, 2 c.c. serum from rabbit No. 66; August 21, twenty-four hours after the last injection the patient had a fainting attack which lasted two or three minutes. There was some redness of the forearm and hand following the injection. August 25, $1\frac{1}{2}$ c.c. serum from rabbit 66; no reaction. August 31, because of the severe reactions, the serum was discontinued, and 2 c.c. beef-lens vaccine was given. September 1, the patient was left in bed after the last treatment and seemed all right. However, 3 hours later, when she got up to go into the dining room, she vomited and on the way back to the ward fainted. September 10, no change was noted in either cataract. September 15, 1 c.c. beef-lens vaccine given; one drop of the vaccine given intradermally showed marked focal reaction. September 20, 25, 30, Oct. 5, 2 c.c. beef-lens vaccine; October 10, 15, $2\frac{1}{2}$ c.c.; October 20, 30, 3 c.c. Examination of the eyes showed but little if any changes in the lenses. One speck of lime deposit was noted on temporal side of the right lens. November 4, $\frac{1}{2}$ c.c. anti-beef-lens sheep serum. November 9, $\frac{1}{2}$ c.c. Blood pressure before injection 190-90; six hours later 194-90. November 14, 1 c.c. anti-beef-lens sheep serum was administered slowly; within 5 minutes, scarlet appearance of body, particularly the face, later a bluish color of the lips, no alteration of circulation or respiration. Because of severe reaction no more injections of serum were given. No changes in cataracts noted on examination. November 19, $\frac{1}{2}$ c.c. beef-lens vaccine; November 24, 1 c.c.; November 29, 2 c.c. Blood pressure before the last injection 220-110; after the injection 180-130, at 4 p. m.; temperature, 102; pulse, 98; at 7 p. m., temperature, 101; pulse, 90. November 30, patient complained of not feeling well but was up and around. December 4, $\frac{1}{2}$ c.c. beef-lens vaccine; no immediate reaction. One hour later temperature, 100; pulse, 80; respiration, 22; six hours later temperature, 98.6; pulse, 80. Because of severe reaction all intravenous injections

were discontinued. December 4, subcutaneous injections of beef-lens vaccines were started. December 9, $\frac{1}{2}$ c.c.; December 14, 1 c.c.; December 19, $1\frac{1}{2}$ c.c.; December 24, 2 c.c. Immediately before injection, blood pressure, 170-90; pulse, 96; six hours after, blood pressure, 180-104; pulse, 90. Differential blood count: No. cells, 212; poly., 70.3; lym., 24.1; l. mon., 3.8; eos., 1.4; bas., 0.4; hem., 78%.

There are practically no changes in the cataractous lenses.

CASE 5.—Louise S., admitted 1918, aged seventy years, white, widowed. Physical condition fairly good; mature, senile, gray cataract right eye, half mature cataract left eye; right anterior chamber normal in depth; projection good; pupils normal. December 13, 1918, successfully extracted the cataract from right eye. September 20, 1921, the left eye has nuclear cataract about two-thirds mature; anterior chamber somewhat shallow, tension normal. The treatment in this case was begun with beef-lens vaccine given intravenously. September 20, 25, 30, 1 c.c.; October 5, $1\frac{1}{2}$ c.c.; October 15, 2 c.c.; October 15, $2\frac{1}{2}$ c.c.; October 20, 3 c.c.; October 30, 3 c.c. November 1, examination of the eye shows no change in the cataract. November 4 began intravenous injections of anti-beef-lens sheep serum, $\frac{1}{2}$ c.c. given as initial dose. Marked reaction within 5 minutes, pain in the back, shortness of breath, tachycardia, tremor, and chilly sensation. Blood pressure, 170-100. Treatment omitted until November 16 when she was again placed on beef-lens vaccine. November 16, $\frac{1}{2}$ c.c.; November 19, $\frac{1}{2}$ c.c.; November 21, 1 c.c. No reaction, pulse, 90; temperature, normal. November 29, 1 c.c. beef-lens vaccine; blood pressure before 160-80, after injection, 160-100. One or two drops of the vaccine given intradermally show no local reaction; that is, there was apparent absence of sensitization. November 30, about 24 hours after the last injection; temperature, 102.6; pulse, 116, intermittent; patient had a chill and complained of general weakness with stomach sickness and headache, but no vomiting. December 4, $\frac{1}{2}$ c.c. beef-lens vaccine; within 3 minutes diffuse redness of the body, chill, severe pain in the back. One hour after the injection, temperature, 99.2; pulse, 86; respiration, 22. Six hours later temperature and pulse same. Because of severe reactions, the vaccines were given subcutaneously. December 9, $\frac{1}{2}$ c.c. beef-lens vaccine; December 14, 1 c.c.; December 19, $1\frac{1}{2}$ c.c.; December 24, 2 c.c. Immediately before injection, blood pressure, 170-90; pulse, 144; six hours after, blood pressure, 180-95; pulse, 120. Marked arrhythmia of the pulse; treatments stopped temporarily. Examination of the eye shows practically no change in cataract. Differential blood count: No. cells, 250; poly., 64.4; lym., 32.8; l. mon., 2.4; eos., 0.4.

CASE 6.—Caroline R., admitted July 11, 1919, aged forty-two, white, married. Physical condition good; varicose veins, both legs; myopia with diffuse nuclear cataracts each eye; the inner cortical layers involved; pupils normal in size and reactions; anterior chamber slightly shallowed each eye. July 26, began intravenous injections anti-human-lens rabbit serum. July

26, 1 c.c. serum from rabbit No. 65. July 27, $1\frac{1}{2}$ c.c. serum from rabbit No. 65. August 5, 10, 15, 20, 25, 31; September 5, 10, 2 c.c. serum from rabbit No. 65. September 15, 1 c.c. serum from rabbit No. 65, one drop injected intradermally; local reaction followed. September 20, 2 c.c. serum from rabbit No. 65; September 25, 30; October 5, 10, 15, $2\frac{1}{2}$ c.c. from rabbit No. 65. November 1, examination of the eyes shows no distinct change in cataracts. November 4, intravenous injections of anti-beef-lens sheep serum begun. November 4, $\frac{1}{2}$ c.c.; November 9, 1 c.c. Blood pressure before injection, 118-70; six hours later, 100-70. November 14, $1\frac{1}{2}$ c.c. anti-beef-lens sheep serum. November 19, $1\frac{1}{2}$ c.c. November 24, 10 A. M., patient complained of headaches; pulse, 102; 10.15 A. M., gave 2 c.c. sheep serum, followed by moderate reaction, pain over the sacrum and down the anterior parts of the thighs, lasting for a minute, moderate redness of the body, no reduction pulse rate; temperature normal. November 29, 2 c.c. anti-beef-lens sheep serum. Blood pressure before injection, 100-70; immediately after, 100-60. Two drops of serum given intradermally showed moderate local reaction. November 30, twenty-four hours after the last injection, temperature, 100.2° ; pulse, 96; complained of pain in the back, not excessive. December 4, $1\frac{1}{2}$ c.c. anti-beef-lens sheep serum; no immediate reaction, but complains of more or less headache all the time. Six hours later temperature 100.6° ; pulse, 90; respiration, 24. Because of the continued reaction the injections were given subcutaneously. December 9, $1\frac{1}{2}$ c.c.; December 14, 2 c.c.; December 19, $2\frac{1}{2}$ c.c.; December 24, 3 c.c. Immediately before the last injection, blood pressure, 100-60; pulse, 90; six hours later, blood pressure, 118-70; pulse, 90. Examination of the eyes shows slight clearing of the lens opacities, especially of the cortical part of the lens. Differential blood count: No. cells, 220; poly., 72.7; lym., 20.9; l. mon., 2.7; eos., 3.2; bas., 0.5; hem., 68 %.

CASE 7.—Jacob L., admitted September 22, 1906. Now aged fifty-six, single, a middle-sized, defective looking individual; diminished knee-reflex action, but no other physical abnormalities. On April 9, 1920, I extracted mature senile gray cataract from right, securing 20/15 V. with plus 10 D. spherical glass (patient coöperative). Vision left eye = 20/15 without glass. February 1, 1921, there is an incipient cataract in left eye; spiculae of opacity at the extreme periphery of the lens, which do not interfere with the vision, which is still 20/15, as it was a year ago. Anterior chamber normal in depth; pupil normal in size and reaction. February 1, 1921, intravenous injections of anti-human-lens rabbit serum were begun (he being the second patient to receive the treatment). February 1, 6, 3 c.c. from rabbit No. 52; February 11, 6 c.c. serum from rabbit 52; February 15, 20, 25, March 2, 7, 5 c.c. from rabbit 53; March 13, 6 c.c. serum from rabbit 59. There were no reactions at all from these injections, but the patient steadily lost in weight. Examination of the eyes showed no change whatsoever in the opacities in the lens and vision remained 20/15. Because of loss of weight

the injections were discontinued for two months, then taken up again in July, but were again discontinued because of steady loss of weight. This patient was examined last on December 24, 1921, when the opacities in the left lens remained exactly as they were at the beginning of treatment, and vision was 20/15. Whether the treatment had anything to do with preventing advance in the opacities in the lens of the left eye is problematic. All we know is the opacities did not increase, though he had a mature cataract develop in the right eye previous to the treatment. Examination of this patient's blood on December 24, 1921, resulted as follows: No. cells, 214; poly., 60.8; lym., 33.1; l. mon., 4.7; eos., 0.9; bas., 1.4; hem., 0.78 %.

CASE 8.—Michael B., admitted October 18, 1921, aged sixty years. Italian laborer, tall, well-developed man, some hardening of the arteries and a moderate arcus senilis each eye, incipient cataract each eye, more marked in right. V. R. E., 20/30 +; L. E., 20/30. Patient was given only intravenous injections of beef-lens vaccines. November 4, 1 c.c.; November 9, 2 c.c.; November 14, 3 c.c.; November 19, 4 c.c.; November 24, 5 c.c. Immediately following the last injection the patient's entire body became flushed, and he complained of nausea and headache. Later, temperature 100; pulse, 90; respiration, 22. November 19, 1½ c.c. beef-lens vaccine; one or two drops of the vaccine were given intradermally; no local reaction. December 4, 1 c.c. beef-lens vaccine; because of reactions, the vaccine from this date was given subcutaneously. December 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately preceding the last injection blood pressure, 130–80; pulse, 80. Six hours later blood pressure, 138–92; pulse, 92. December 24 examination of eyes shows no change in the lenses, vision remains as at beginning of treatment, six weeks ago. Differential blood count: No. cells, 246; poly., 57; lym., 29.7; l. mon., 10.9; eos., 2; bas., 4; hem., 59%.

CASE 9.—Bernadino C., admitted August 9, 1921, aged sixty-nine years. Italian, male, white. Physically undersized, well-preserved old man, showing moderate general arteriosclerosis, chronic valvular endocarditis. Pupils normal in size but somewhat sluggish in reaction. Incipient cataract in each eye, more marked in right, anterior chambers, normal depth. Intravenous injections begun September 10, 1 c.c. beef-lens vaccine; September 15, 1 c.c. beef-lens vaccine; September 20, 1 c.c. anti-human-lens rabbit serum; September 25, 2 c.c. anti-human-lens rabbit serum; September 30, 3 c.c. anti-human-lens rabbit serum; October 5, 4 c.c. anti-human-lens rabbit serum. No reactions from treatment. October 10, 2 c.c. beef-lens vaccine; October 15, 20, 25, 30, 5 c.c. beef-lens vaccine. No change observed in the cataracts. November 4, 1 c.c.; November 9, 2 c.c.; November 14, 3 c.c., and November 19, 4 c.c. anti-beef-lens sheep serum. Following this last injection patient developed temperature, 100; pulse, 80; respiration, 22, and 24 hours later a severe skin eruption which covered the entire body; there were large red, raised spots which looked like a severe case of hives.

November 24, 5 c.c. anti-beef-lens sheep serum. Patient complained of severe headache and pains in his back and legs; temperature, 99. Because of reactions, the serum from December 4 was given subcutaneously. December 4, 1 c.c. anti-beef-lens sheep serum; December 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately before this last injection, blood pressure, 118-76; pulse, 76; after, blood pressure, 128-70. Differential blood count: No. cells, 214; poly., 35.5; lym., 60.8; l. mon., 3.7; hem., 71%. Examination of the patient's eyes showed no change one way or the other in the lens opacities, seemingly the cataracts had been arrested in development. However, the patient had not been under observation long enough to determine this.

CASE 10.—Sherwood F., admitted December 14, 1918, aged sixty-three years at that date. English, widowed, well nourished and in good physical condition. Right eye has incipient cataract, left eye almost mature gray cataract, anterior chamber slightly shallow each, more marked left, right pupil normal in size and reaction, left slightly irregular. V. R. E., 20/200; L. E., l. p., projection good. Began with intravenous injections of beef-lens vaccine. September 15, 1921, 1 c.c.; September 25, 2 c.c.; September 30, 3 c.c.; October 5, 4 c.c.; October 10, 2 c.c.; October 15, 20, 25, 30, 5 c.c. November 1, no change in the cataracts. November 4, 1 c.c. anti-beef-lens sheep serum; November 9, 2 c.c. serum; November 14, 3 c.c. serum. Examination of the eyes shows slight increase of vision in each. R. E., 20/70; L. E., 1/200. November 19, 4 c.c. anti-beef-lens sheep serum. Following this injection there was a general flushing of the skin of the whole body, temperature, 100.6; pulse, 96; respiration, 26. November 24, 5 c.c., and November 29, 1 c.c. anti-beef-lens sheep serum; one or two drops of the serum introduced intradermally gave decided local reaction (positive sensitization test). Because of the reaction injections were given subcutaneously. December 4, 1 c.c.; December 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately before this last injection, blood pressure, 162-80; pulse, 60; after, blood pressure, 150-110. Blood examination: No. cells, 220; poly., 55; lym., 27.7; l. mon., 5; eos., 10.9; bas., 1.4; hem., 68%. Examination of the eyes December 24 shows slight clearing of lens in each, but not pronounced.

CASE 11.—James S., admitted August 25, 1921, aged fifty years. Irish, horseshoer, well-nourished man, double hernia. Incipient cataract right, half mature left. V. R. E., 20/40—; L. E., 20/200 +. Pupils slightly sluggish to light, anterior chamber normal right, shallow left. September 10, began intravenous injections of beef-lens vaccine. September 10, 15, 20, 1 c.c.; September 25, 2 c.c.; September 30, 3 c.c.; October 5, 4 c.c.; October 10, 2 c.c.; October 15, 20, 25, 30, 5 c.c.; no change in cataracts. November 4, 1 c.c. anti-beef-lens sheep serum; November 9, 2 c.c. serum; November 14, 3 c.c. serum; November 15, clearing of opacities. V. R. E., 20/30; L. E., 20/200. November 19, 4 c.c. anti-beef-lens sheep serum. Following

this injection patient developed temperature, 102.6; pulse, 102; respiration, 22, and showed a general flushing of the skin over the entire body. November 24, 5 c.c., and November 29, 1 c.c. anti-beef-lens sheep serum. Intradermal sensitization test positive. Following the last injection, patient developed urticaria, and complained of pain in back and legs. From this date on injections of the serum were given subcutaneously. December 4, 1 c.c.; December 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately before the last injection, blood-pressure, 102-78; pulse, 64; after, blood pressure, 98-78. Differential blood count: No. cells, 517; poly., 46.4; lym., 41.8; l. mon., 9.5; eos., 1.9; bas., 4; hem., 72%. December 24, eyes in same condition as of November 15.

CASE 12.—Joseph H., admitted August 9, 1921, aged eighty-two years. German, well-developed, well-nourished man, systolic heart murmur, absence knee reflex, epithelioma left side nose. Incipient cataract each eye, more marked left. V. R. E., 20/100; L. E., 20/200. Pupils, right larger than left, both sluggish to light stimulus; good for accommodation. September 10 began intravenous injections of beef-lens vaccine. September 11, 15, 20, 1 c.c.; September 25, 2 c.c.; September 30, 3 c.c.; October 4, 4 c.c.; October 10, 2 c.c.; October 15, 20, 25, 30, 5 c.c.; November 1, no change in the cataracts. November 4, 1 c.c. intravenous injection of anti-beef-lens sheep serum; November 9, 2 c.c. serum; November 14, 3 c.c. serum; November 19, 4 c.c.; November 24, 5 c.c.; November 29, 1 c.c.; December 4, subcutaneous injections of anti-beef-lens sheep serum were begun. December 4, 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately before this last injection, blood pressure, 90-66; pulse, 80; six hours after, blood pressure, 98-70. December 24, differential blood count: No. cells, 253; poly., 63.2; lym., 29.7; l. mon., 6.7; eos., .4; hem., 88%. There was but little change produced in the lens opacities by the treatment and vision was unaltered. The patient had no reactions following the injections either of the vaccines or serum.

CASE 13.—Edward B., admitted in 1901; good physical condition; anophthalmos right; incipient cataract at periphery, inferior nasal quadrant, left, pupil normal in size and reaction. V., 20/20; anterior-chamber normal in depth. September 10, intravenous injections of beef-lens vaccines begun. September 10, 2 c.c.; September 15, 20, 1 c.c.; October 5, 10, 2 c.c.; October 15, 20, 25, 30, 5 c.c.; November 1 no change observed in the cataract. November 4, intravenous injections of anti-beef-lens sheep serum begun. November 4, 1 c.c.; November 9, 2 c.c.; November 14, 3 c.c. V., 20/15, some clearing of opacity in the lens. November 19, 4 c.c.; November 24, 5 c.c., and November 29, 1 c.c. anti-beef-lens sheep serum. Intradermal sensitization test positive. December 4, subcutaneous injections of the sheep serum were begun. December 4, 1 c.c.; December 9, 1 c.c.; December 14, 2 c.c.; December 19, 3 c.c.; December 24, 4 c.c. Immediately before this last injection, blood pressure, 142-90; pulse, 70; after, blood pressure, 172-82.

Differential blood count: No. cells, 127; poly., 73-8; lym., 23; l. mon., 2.3; eos., .9; hem., 67%. Examination of the eye shows little change in lens opacity. V., 20/15.

CASE 14.—This case is of interest, as here the treatment was given intensively, that is, every other day. Mrs. W., aged seventy-five years; a mature senile gray cataract in the right eye. The left lens had been successfully extracted twelve years previously. Anti-beef-lens sheep serum was begun February 17, 1922, intravenously, the initial dose being $\frac{1}{2}$ c.c., which was increased $\frac{1}{2}$ c.c. each succeeding dose. The patient got general reaction after the sixth dose, which was $3\frac{1}{2}$ c.c. of the serum. March 17, 1922, one month after the injections were started, there were marked changes in the crystalline lens. Numerous wedge-shaped sectors had become semitrans-

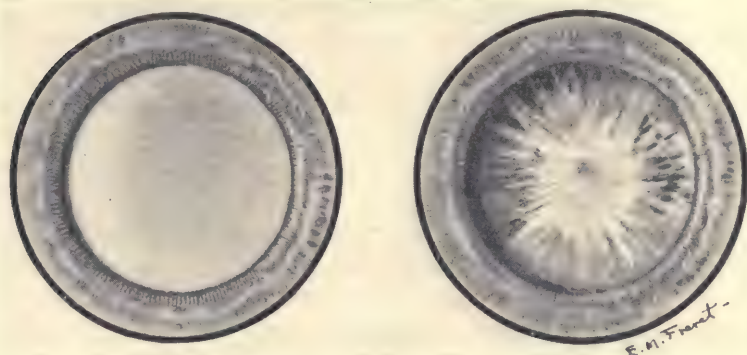


Fig. 2.—Case 14. Mrs. W. R. E.; two stages.

parent; a great part of the anterior cortical layers was absorbed, so that a distinct shadow was cast on the nucleus of the lens by the iris when light was focused obliquely into the pupillary space. One month later the effect was even more pronounced (Fig. 2). In fact, more effect was produced on the lens of this patient in eight weeks of intensive treatment than in any of the other cases, some of whom have been under treatment for fifteen months. It would seem, therefore, that a quicker and more effective result is obtained by intensive treatment. Guided by the result in this case, just as soon as the serum can be had in quantity, which the manufacturers, H. K. Mulford & Co., promise in the near future, I intend to start the treatment with a massive intravenous injection, 50 to 100 c.c., then follow it up with injections of the vaccine every other day. In this way it is hoped to obtain a very decisive result and one brought about in a reasonably short time—a few weeks.

COMMENTS

1. It will be noted in these experiments that both sera and vaccines have been used, and in some patients both the sera and vaccines at different periods of the treatment; also at the beginning of the experiments that only the human cataractous lenses were used as vaccines, and as antigen to produce anti-lens sera. Although it was known that the lens antigen was organ-specific in its action, nevertheless it was thought that lens antigen from the same species would possibly produce a more powerful serum than lens antigen from a foreign species. Accordingly at first only the human lens antigen was used. Subsequently, because of the difficulty in securing sufficient human lens, beef lens antigen was used in the preparation of anti-lens sera. The reason for changing from anti-lens sera to lens vaccines in the same patient was twofold: first, the intravenous injection of serum, when increased up to 5 c.c., and sometimes much less quantity in female patients, caused marked anaphylactic shock; second, it was thought perhaps that the *active* stimulation of lens antibodies in the patient, by injections of vaccines, would have a more lytic or dissolving action than the *passive* treatment by means of the small amount of lens antibodies contained in the serum injected.

It was found by actual experience, however, that the lens vaccines also produced shock when given intravenously and carried up to as high as 5 c.c., or even less quantity. Because of this shock effect, which at times was quite alarming, both with the sera and vaccines when given intravenously, we finally decided to give all injections, sera and vaccines, subcutaneously. Since this method was adopted, no symptoms of shock so far have followed, though as much as 5 c.c. sera or vaccines have been given at one dose. We advise, therefore, for safety's sake, that these remedies be thus given, except by those who are quite familiar with this method of treatment.

2. *Source and Strength of the Sera.*—Since the lens is organ-specific and not species-specific, the question of securing sufficient lens antigen to manufacture anti-lens serum has been greatly simplified, as beef, sheep, and swine lenses can be had in any quantity; and sheep, cows or horses even, can be used as the source of the sensitized serum. In our experiments, instead of human lenses as the antigen, and rabbits as the source of the sensitized serum, we used beef lenses as the antigen and a sheep as the source of the sensitized serum. This is a practical

method, and if the treatment ultimately proves successful, there will be no lack of remedy with which to treat patients.

3. *Standardization of the Sera.*—So far we have not been able to have a standardized serum, but trust to have this later. Guyer and Smith state that in their experiments, inasmuch as they had no visible way to tell when serum is adequately sensitized for use as a cytolyisin beyond trying it out directly, a series of lens precipitin tests were made with various of the fowls (which they used as the source of sensitized serum) after the fifth and sixth injections of antigen, respectively, in order to be sure they were responding to the lens proteins. They do not claim a necessary connection between the precipitin and cytolyisin reactions of the blood, but it is thought that, if the lens had so sensitized the fowls that precipitins were formed, one might infer that cytolyisins had also been generated. It is possible, therefore, and feasible to test the animals—(rabbits, sheep, etc.) we are now using as the source of the anti-lens sera, in same manner to determine if the sera are strongly sensitized with cytolytic substances.

4. *General Reactions.*—First to be considered is shock. Several of our cases suffered more or less severely from shock, both with the sera and vaccines, at times even when small doses were administered intravenously. The whole surface of the body within 60 seconds would turn intensely red, then after a few moments would become more or less livid, labored and irregular breathing with great dyspnea, rapid weak pulse, which at times was quite irregular, great restlessness, at first quickly followed by marked general weakness and in the severe cases shock; occasionally there was involuntary movement of the bladder and bowels. Perhaps the most distressing symptom was that of impending suffocation. The blood pressure following shock varied; in most of the cases it was lowered; however, in others it was elevated. As regards temperature, unfortunately this was taken in our cases in but a few instances. In animals, usually, there is a marked drop in the temperature following shock, and Pfeiffer¹ states that by this symptom alone the mildest anaphylactic reactions may be detected even when other symptoms are not present.

The second general effect of the prolonged administration of anti-lens sera and vaccines is that of anemia, more or less pronounced. In one of our cases (Case 7), the second to have the treatment (which extended over a period of about 6 months), the patient became pale,

¹ Pfeiffer u. Mita: Zeits. f. Immunitätsforschung, 1910, iv.

lost weight, and appeared to be "drying up," as one of the staff at the hospital remarked. He never suffered shock, or had any other ill effect from the injections, except that he gradually lost weight without any apparent cause, and for this reason alone the injections were discontinued. This patient's differential blood count was not tested until December 24, 1921, when the hemoglobin was 78 per cent.

A third general symptom that appeared in one case (Case 9) was an extensive urticaria, manifesting itself as large wheals, an inch or more in diameter, elevated above the surrounding skin surface, and covering most of the body, which lasted a few days. No other patient was similarly affected. All of the patients treated, except one or two, responded to the sensitization tests when a small amount of the serum or vaccine, with which they were being treated, was injected intradermally.

5. *Local Reactions.*—How and why the antibodies contained in sensitized sera, or the antibodies produced by the injection of the vaccines directly into the animal, act selectively and specifically on the tissues used as antigen are still conjectural. All we know definitely is that they have a more or less specific action, the sera acting passively and the vaccines actively. Guyer and Smith¹ in their extended experiments on pregnant rabbits and mice with fowl serum sensitized to rabbit lens and mice lens, respectively, have noted opacification and liquefaction of the lenses of the young, but never in the mothers, who were directly injected. In the case of one young mouse, the lens in one eye was rendered opaque, and in the other eye, was liquefied. In no instance were the lenses of the mothers affected. Their comment relative to this point is as follows: "In so far as the literature on cytolytins records positive results, it leads one to expect specific effects in the immediate animal injected; a possible explanation of the lack of effect on the mother may be that because of the meager circulation of blood in the lenses of adults, the quantity of cytolytic serum which reaches a lens is insufficient to affect it."

On the lenses of the developing young *in utero*, as is well known, there is a vascular membrane, and the cytolytic substance can reach these lenses and have a much greater effect. Furthermore, such developing lenses are softer and have not the fibrous masses as obtains in older animals. Guyer and Smith attribute the *liquefaction* of lenses observed by them in the young of rabbits and guinea-pigs to a true

¹ Guyer and Smith: Jour. Experimental Zoölogy, May, 1918, p. 681.

cytolytic action of the sensitized serum used by them. Whether the clouding and opaquing which occurred in other of the lenses should be regarded as the result of a cytolsin or of a precipitin, they regard as problematical.

A further possible explanation of why cytolytic sera do not affect the lenses of adult animals or man powerfully, may be due to the lack of alexin or complement in the aqueous humor of the anterior chamber of the eye. This curious fact is called attention to by Zinsser,¹ who cites the experiments of Metchnikoff: "In this fluid no alexin is present under normal conditions, but if puncture is practised and the fluid again taken after a period of two or three hours, alexin is now found, probably according to Metchnikoff's school, because of the coincident entrance of leukocytes into this space. It is conceivable, however, that the aqueous humor may be free from alexin for other reasons than the absence of leukocytes; and an injury followed by the invasion of leukocytes is pretty sure to be followed also by the entrance of the fluid elements of the blood, *i. e.*, alexin."

This fact would suggest one practical point in the attempt to absorb or liquefy cataracts in the human animal, that is, during the period that the vaccines are being given, the eyeball should be massaged, or subconjunctival injections of some slightly irritating fluid be given. In fact, any safe means employed that would induce an increased flow of the plasma and along with it leukocytes into the anterior chamber of the eye; because it is absolutely necessary to have complement or alexin present in conjunction with the antibodies of the sensitized serum in order to obtain a lytic effect—that is, absorption or liquefaction of the lens.

6. The local effects of sera and vaccines on the cataractous lenses, that is, as regards mature and immature cataracts, were exactly the opposite to what we had preconceived at the beginning of the treatments. We had supposed that the immature cataracts with the scattering or partial opacities would be most easily affected. It proved to be exactly the contrary. The only two patients in whom we obtained partial liquefaction of the lenses were those having mature, gray, senile cataracts, where a great part of the cortical layers was absorbed. The sera and vaccines seemed to have no liquefying effect on the spiculæ in the incipient cases, but they did seem to exert a retarding effect on their progress, that is, they seemed to prevent further develop-

¹ Zinsser: *Loc. cit.*, p. 171.

ment of the cataracts. This was obtained particularly in case No. 7. We know, however, that incipient cataracts may remain stationary for years, so a larger number of cases must be treated and a more extended time allowed to ascertain if the serum actually arrests development of immature cataracts. The one case of sclerosing black cataract was not affected in the least by the treatment. Perhaps a possible explanation of the more marked effect on mature senile cataracts is that the fibers are more or less disintegrated, and the broken down fibers, for that reason, can be more easily liquefied.

Perhaps more rapid and satisfactory effects could be obtained if the patients received more intensive treatment, that is, to give the injections every other day for two weeks, then rest a week, and again repeat the injections for two weeks. However, this remains for the future.

7. It may be asked by some, why should these insane patients blinded by cataracts be subjected to more or less risky treatment? For two reasons: first, many of the patients cannot be operated upon for the cataract because of their disturbed condition, the patient destroying the eye at the time of the operation, or subsequently, before the wound is healed, by his violent actions. No such risk to the eye is incurred when vaccines and immunized sera are given; second, when these anti-lens sera and lens vaccines are given subcutaneously or deep into the tissue, no more risk is incurred to the patient's general condition than is now taken daily by injecting the various bacterial vaccines and sera, for instance, diphtheria antitoxin. The general condition of these insane patients is often greatly improved by the removal of cataracts; therefore, if we can devise a safe means for the absorption of their cataracts, even in the most disturbed cases, we will have accomplished a great advance in their treatment. Furthermore, the full consent of relatives or friends was obtained before these experiments were started.

8. Twelve of these patients were bled on December 24, 1921, and precipitin tests on their sera were made, which proved rather inconclusive, as serum in Case No. 2 was negative in all the dilutions of the serum. On the other hand, in Case No. 3 the test was positive in dilution 1:10; in Case No. 9 + in dilution, 1:100, and in Case No. 11, + in dilution 1:100. It will be noted in case reports that the lenses in Cases 3 and 9 were not affected by the treatment, while in Case 11 only slight effect was produced on the lens.

PRECIPITIN TESTS ON SERA OF PATIENTS TREATED WITH LENS ANTIGEN

No. of Case	Dilution of Lens Antigen	Dilution of Serum		
		1:10	1:100	1:200
1	1:10	1:10	1:100	1:200
2		—	—	—
3		+	—	—
4		—	—	—
5		—	—	—
6		—	—	—
7		—	—	—
8		—	—	—
9		—	+	—
10		—	—	—
11		—	+	—
12		—	—	—
13		—	—	—

CONCLUSION

No definite conclusions can be drawn from so few cases (13) as here reported. The partial results so far obtained would seem to indicate: First, that mature senile cataracts (at least the cortical layers) may be absorbed or liquefied; second, a possibility that immature cataracts may be retarded or entirely checked in their progress. The present paper is to be considered simply as a preliminary report, to be followed by further and more extended experiments.

I cannot close the paper without expressing my deep appreciation of the assistance given me by Dr. Russell L. Cecil, who prepared the sera and vaccines and gave valuable suggestions in the method of administration, and my thanks are due to Dr. George A. Smith, Superintendent of the State Hospital at Central Islip, N. Y., and to the staff of this institution. I am particularly indebted to Dr. Ralph G. Reed, Dr. Charles Vaux and Dr. McNeill, who helped conduct the treatment and who gave many of the injections. Without their assistance the treatment could not have been carried on. The pathologist of the institution, Dr. King, made the differential blood counts and hemoglobin tests.

DISCUSSION

DR. LUCIEN HOWE (Buffalo, N. Y.): In dealing with this question, it seems essential to remember that the lens is a complex substance. The chemists tell us it contains at least five different proteins. What the action of one may be as compared with another as yet we know not.

It is quite sure, however, that in one way the lens does act as a lysin. That

is shown by the experiments of Guyer. But this action may prove to be a two-edged sword. Although we may sometimes obtain action on the lens which we wish, we may also get a severe anaphylactic action otherwise, and destroy other parts of the eye. This is a new subject, one that gives us hope for the future. But at present the only patients who will consent to be treated in that way, according to the doctor's report, are those who are already in an insane hospital. All that can be said is that for the present the curtain is drawn aside and we get a glimpse of the great field for investigation that lies beyond.

PROF. M. F. GUYER (Madison, Wis.): I know nothing about the clinical or the pathologic aspects of this problem. I am a mere biologist, but I have been interested in Dr. Davis' report, particularly in connection with his use of vaccines. In our own case, as he has said, we observed no direct effect on the eyes of the injected mother rabbits. But, as a matter of fact, we judged largely by obvious defects. We have not tried to go into the refinements of the question; we have gone largely by conspicuous marks like a cataractous lens.

In our experience defects were produced first in the uterine young. We timed our injections so as to get them into the pregnant mother about the time the lens was forming at its best in the young, which means about the tenth to the fourteenth day. It is surrounded then by a network of blood-vessels, and anything we got through the placenta could easily reach the lens by means of the circulating blood. Lens-defects established in this way apparently became hereditary.

The thing that has interested me most in Dr. Davis' account is that he gets direct effect on the mother. And the paper of Drs. Verhoeff and LeMoine also speaks of the fact that where you have an injured lens you are likely to get sensitization. This suggests that a lytic substance may have been produced in the blood stream; and may not such lytic substances operate on the cells of the young in pregnant females so as to form the beginning of congenital defects? Certainly in the eyes of some of our fetal rabbits we secured such an effect. This occurred not only after the injection of fowl-serum immunized against rabbit lens, but also when we injected rabbit-lens directly into pregnant rabbits. It is difficult to get the defect in the latter way. One female so treated, however, has three defective-eyed young. This opens up the whole field of congenital defects in the first place. If an animal can build up antibodies against its own tissues when these become injured or diseased, and if such antibodies can also affect the fetus or the gonads, you see where the facts lead. Our work with rabbits tends to make us believe that there is some thread of chemical identity between substances in the germ-plasm and the finished organ in the adult. And if you get a lytic effect in one may you not get a like action in the other?

DR. HENRY H. TYSON (New York City): Clinically, the subject of Dr. Davis' paper is one of interest not only to ophthalmologists but to those who may be afflicted with cataracts, for individuals if given their choice between medical and surgical treatment, with anticipated equal results, will in a vast majority of cases select the former procedure. This being so, it can be

readily seen how important these preliminary researches may be, providing the future should fulfil Dr. Davis' fondest hopes.

Unfortunately, in my opinion, the results so far obtained and reported are too few and of too short duration to enable us to form reliable conclusions. All of us have seen quite similar results in cases, some with no treatment and others under what might be termed ordinary routine tonic treatment.

Guyer and Smith, in their researches in animal experimentation, demonstrated the fact that while lens serum did not apparently affect the eyes of the adult animal injected, it did produce anatomic changes in the eyes of the offspring. Dr. Davis, however, with his method thinks he finds that the cortex of the cataractous lenses of human adults is acted upon with similar serums and vaccines. Assuming this to be a fact, much progress still remains to be made in finding a solvent for the nucleus of the lens before it could assume a definite, practical therapeutic value.

That spontaneous absorption of cataractous lenses may occur I can testify from a recent personal observation of an unrecorded case in a physician aged forty years, with soft cataract and an uveitis anterior, who, while under treatment with thyroid extract and tuberculin therapy for the latter condition, had his cataractous lens absorbed, leaving only the capsule remaining. There was no history nor evidence of traumatism while under observation.

If Dr. Davis' future researches should prove that he can at will aid or induce nature to produce results similar to the foregoing case with his method of vaccine and serum therapy, he will then have added a very important and valuable chapter to the history of "bloodless surgery of the eye."

DR. FREDERICK A. DAVIS (Madison, Wisconsin): What I have to say has only an indirect bearing on the subject, as it deals exclusively with rabbits in which hereditary blindness has been produced by the injection of fowl serum immunized against rabbit lens, or by direct injection of lens into the pregnant mother, after the method of Dr. Guyer.

Recently I have undertaken, with my associate, Dr. Neff, the study of all the defective-eyed stock in Dr. Guyer's laboratory. To date we have studied about 20 pairs of eyes. The work is not yet finished, and therefore only an incomplete report can be given at this time. Of the rabbits studied so far which showed some abnormality, six had one normal eye. The right eye was more frequently affected than the left. The most striking and more typical changes are as follows:

The globes varied in size and shape from an almost total absence to a size somewhat larger than normal, the atrophic and microphthalmic globes predominating. The tension to palpation was usually subnormal. The conjunctivæ frequently were watery and injected. The sclera often showed marked thinning between the muscle attachments, with a staphyloma in the posterior and inferior portion of the globe. This was so great at times that the cornea became displaced behind the lids, the large staphylomatous mass protruding between them.

The corneæ were at times clear, though frequently a keratitis was present, especially characterized by an invasion of the conjunctiva at the limbus, or an appearance resembling sclerosing keratitis. The central portion of the

cornea often showed a deep opacity. The anterior chamber varied in depth, some being deep, some shallow. They were usually deep where the globe was not atrophic.

The iris, with one exception, showed a large coloboma below. They appeared atrophic and usually did not react to light. Abnormal vascularization was present in some instances.

The lens usually showed some form of opacity, varying from discrete cortical spots to a general diffuse cloudiness, and at times an appearance of calcareous degeneration. One post-polar cataract was observed. The lenses appeared to be dislocated below and backward in the colobomatous area. No liquefaction or absorption of the lens has yet been found in this series.

The vitreous was usually apparently clear in the upper portions of the globe, but in the lower half it was filled with masses of vascularized tissue or exudate, especially in the region of the coloboma. Numerous blood-vessels could be seen in these masses, and they often appeared to run forward in an anteroposterior direction.

The choroid practically always showed a broad coloboma below, spreading from the nerve-head through the ciliary body. The normal arrangement of the choroidal vessels could be made out above the disc with the ophthalmoscope. The margins of the coloboma were often clear-cut and sharply defined, but a clear view of the entire colobomatous area was obscured by the vascularized masses of tissue anterior to it.

The optic nerve head and retina in the normal eyes showed the characteristic arrangement of the medullated nerve-fibers, with an unusually deep cupping of the entire disc, varying in depth from 4 D. to 9 D. In the diseased eyes only the upper margins of the disc could be made out, the lower portions merging with the coloboma of the choroid below. We have not yet determined whether or not there is a coloboma of the retina and optic nerve. There was a rather striking absence of hemorrhages except in one baby rabbit. The retinae were apparently normal in the upper part of the eye.

It is interesting to note that these more or less typical changes were already present in the eyes of a baby rabbit of two months, a specimen of which I have with me.

DR. A. E. DAVIS (closing): I realize the two-edged sword that Dr. Howe spoke of. Some years ago I consulted with Dr. Flexner, of the Rockefeller Institute, and he also mentioned the same point. In fact, that is one of the reasons I gave up the experiments in the early part of them. However, after I had seen the results of Guyer and Smith I did not hesitate to go on. As Dr. Howe has said, the most of them were on insane patients. I may say, however, that I have now five private patients who have come and asked to be treated. It is a rare thing when a patient comes in with cataract and wants to be operated. Like the old colored gentleman down South, they usually ask if you cannot "scatter" it. We hope to make the operation for cataract obsolete.

FACOERISIS

PROF. IGNACIO BARRAQUER

Barcelona, España

Daviel, al idear la extracción de la catarata, se proponía hacer la extracción total, pero dificultades técnicas le impidieron llevar á cabo su deseo, quedando instituido desde entonces el método clásico por quistitomia, que se ha practicado con muchas modificaciones en su técnica, todas ellas encaminadas á extraer la mayor cantidad posible de restos blandos y cápsula del cristalino, con el minimum de maniobras. Prueba evidente del profundo convencimiento, que en el ánimo de todos los oftalmólogos existe de que la mayor parte de accidentes operatorios son debidos á las múltiples maniobras, que requiere una evacuación completa de masas (presiones, lavados, iridectomia, etc.), y de que los accidentes post-operatorios reconocen por origen las flógosis determinadas por dichos restos y los enclavamientos de los mismos.

A pesar de todas las modificaciones llevadas á cabo en la quistitomia y de las operaciones complementarias ideadas (iridectomia preparatoria, discisión, etc.), para obtener los resultados de una operación perfecta, nunca con dicho método se puede lograr este ideal, toda vez que siempre dentro del ojo quedan restos de la lesión que nos proponemos extirpar.

La extracción simple total es el único método racional que existe para operar la catarata. Esta se ha practicado por muchos procedimientos que, si bien libraban al ojo de los accidentes post-operatorios, por dejarle libre de restos de catarata, le predisponían á accidentes operatorios, por complicar las maniobras del acto quirúrgico.

Convencido de que únicamente debíamos practicar la extracción total, ensayé la mayor parte de los métodos existentes durante varios años, cerciorándome de que los menos expuestos eran los que, cogiendo el cristalino por su cara anterior, lo extraían fuera del ojo, envuelto en su cápsula, si bien los instrumentos para ello ideados rasgaban la cápsula cristaliniiana, convirtiendo en la mayoría de casos la tan deseada extracción total en una simple quistiectomia; era pues pre-

ciso construir una pinza que no rompiera la cápsula del cristalino. Esta fué mi idea primordial del procedimiento que denominé Faco-érisis, del griego Faco, lente y Erios, arrancar. Una pequeña ventosa adaptada á la cara anterior del cristalino debía hacer presa fuertemente en él y permitir arrancarlo, rompiendo las débiles fibras de la zónula que le mantienen en su sitio. Muchas fueron las tentativas y pruebas, que llevé á cabo variando la forma y dimensiones de la ventosa y la intensidad del vacio; antes de lograr, coger y sacar enteras las cataratas con un minimum aceptable de rupturas de su cápsula pero sin embargo las tracciones que sobre la inserción periférica de las fibras zonulares se practicaban, predisponían al ojo á complicaciones por parte de la región ciliar. Mi método, distaba mucho de la perfección, si bien obtenía con él resultados superiores á los de la quistitomia.

Mi primer Erisifaco era una pinza pneumática, mientras que el actual es pinza pneumática y zonulotomo toda vez que al coger el cristalino y deformarle, reduciendo su diámetro máximo, lo hace por pequeñas y frecuentes sacudidas, con las que se le imprime un movimiento vibratorio rapidísimo, y de suficiente altura de onda, para que al ser transmitido á las fibras zonulares ocasionen su ruptura cerca del cristalino, sin que la fuerza tenga tiempo de transmitirse á su inserción periférica, y sin que la intensidad de vacio empleada sea capaz de romper la elasticidad de la cápsula, rompiéndola. Claro está que dicha intensidad de vacio (altura de la vibración) y el número de interrupciones por minuto (amplitud de la vibración) son condiciones mecánicas del enrarecimiento del aire en la ventosa, que deben guardar relación con las condiciones físicas de cada uno de los ojos á operar, y que el operador debe regular de antemano en su Erisifaco, con arreglo á la mayor o menor dureza del cristalino y esclerosis de la zónula.

Las alturas de onda (intensidad de vacio), que empleamos, están en razón directa de la mayor dureza del cristalino, y la amplitud de las vibraciones (número de las mismas) en razón inversa de la mayor esclerosis de las fibras, de modo que en una catarata incompleta, blanda, deformable, con una zónula elástica poco esclerodada, debemos emplear un vacio débil y un número de vibraciones grandes, mientras que en los sujetos viejos con cataratas completas y zónulas friables, hace falta mayor intensidad de vacio para deformar el cristalino y siendo menos elástica su zónula, necesita menor número de vibraciones para romperse.

A pesar de haber logrado la extracción sin tracciones en la inserción periférica de la zónula, se veían mis estadísticas afectadas por alguna iridociclitis tardía, iridociclitis debida al enclavamiento en los labios de la herida de las fibras zonulares o de los ángulos del coloboma quirúrgico. Complicación que raramente se presentaba en los casos operados sin iridectomia, por lo que, salvo en casos excepcionales, hago la extracción simple con una pequeña brecha periférica en el iris para evitar la hernia.

Las maniobras operatorias en una Facoerisis simple, quedan reducidas á la mas mínima expresión, pues que una vez tallado el colgajo, basta introducir en la cámara anterior un solo instrumento, el Erisifaco, que aplicado sobre la cara anterior del cristalino adhiere á ella y lo libra de sus naturales adherencias, sin que el ojo se deforme, sin la menor violencia, y con solo retirar el instrumento de la cámara anterior, con suavidad pasmosa se ve salir el cristalino envuelto en su cápsula, adherido á su extremidad quedando la pupila de un negro purísimo, central y redonda.

Las reacciones post-inflamatorias debidas en la quistitomia: 1° á las repetidas maniobras del acto quirúrgico, 2° á los restos de substancias degeneradas del cristalino y su cápsula, y 3° á los enclavamientos de restos de la cápsula entre los labios de la herida, no existen, toda vez que con una sola maniobra extirpamos de una vez toda la lesión, sin dejar dentro del ojo el menor resto de ella.

La presencia de restos cristalinianos en el humor acuoso y la flogosis que ellos originan, convierten á la cámara anterior en un terreno abonado á los gérmenes infectivos, condiciones que no existen en la extracción total, por lo que las infecciones en esta clase de operación son rarísimas.

La técnica de la operación es fácil, pero requiere una serie de pequeños detalles indispensables para el buen resultado. Los enfermos son examinados detenidamente, con la pupila dilatada, al microscopio binocular y lámpara de hendidura, á fin de conocer las características del cristalino, su cápsula, y la zónula, que nos han de permitir graduar la intensidad del vacío y el número de vibraciones por minuto, con que debe llevarse á cabo la intervención, así como tambien el tiempo necesario para obtener la dilatación pupilar deseada y asesorarnos de que el ojo reúne las condiciones de asepsia necesarias para sufrir una intervención.

Prevía dilatación pupilar por un midriático de acción fugaz (eufatmina), tallamos con el cuchillo de Graeffe un colgajo de los $\frac{2}{3}$ de la

circunferencia de la córnea, emplazando en ella sus dos tercios estromos y el tercio medio en la esclerótica, acabándolo con una larga lengüeta conjuntival, en la que, si lo creemos indicado, emplazamos un punto de sutura que anudaremos después de terminada la operación. Si ésta debe ser simple, introducimos en la cámara anterior, rozando la cara posterior de la córnea, sin tocar el iris, la pequeña ventosa del Erisifaco, previamente regulado con arreglo á las condiciones físicas del ojo á operar, y al nivel del centro de la pupila se aplica sobre la cristaloides sin ejercer la menor presión, y la hacemos resbalar hacia abajo hasta que su extremo inferior se oculta debajo del iris, en la cámara posterior; entonces es cuando abriendo paso al vacío, se produce el enrarecimiento del aire en la ventosa á la que se adhiere el cristalino, rompiéndose instantáneamente las fibras de la zónula. Con suma lentitud y sin hacer nunca la menor presión sobre el vítreo, hacemos que el cristalino gire sobre un eje transversal hasta que su borde inferior, rozando la cara posterior del iris primero y la de la córnea después, se convierta en superior en cuyo momento, con solo retirar el instrumento del ojo acabamos la extracción, quedando la toilette reducida á la aplicación de la pomada de eserina para que el miosis producido prive el enclavamiento de las fibras zonulares.

Si la talla del colgajo y la maniobra de extracción han sido efectuadas con suavidad y sin hacer la menor presión sobre el globo ocular, nunca tendremos pérdidas de vítreo, ni accidentes operatorios, á no ser que el paciente contraiga el orbicular de los párpados o haga movimientos intempestivos. Para evitar con toda seguridad los movimientos que por la indocilidad del paciente pudieran sobrevenir, provocamos como Van Lint y Villard la parálisis temporal del orbicular.

Aunque soy partidario entusiasta de la extracción simple, practico en casi todos mis enfermos un pequeño agujero en la periferia del iris, que sin privarnos de las ventajas de aquella, nos da las garantías de la iridectomia en cuanto á la hernia del iris.

Regulando apropiadamente la intensidad de vacío y número de vibraciones, pueden extirparse sin temor á accidente alguno, toda clase de cataratas (maduras, incipientes, Morganianas, etc.) en sujetos mayores de 40 años de edad, y en sujetos mas jóvenes todas las completas y aun algunas variedades de las incompletas en que la zónula se presenta muy esclerosada. Las cataratas polares posteriores sintomáticas de la miopia, se extraen con facilidad ya que la zónula en ellas es muy frágil.

Llevo practicadas más de 2800 Facoerisis y los únicos accidentes operatorios que en estos últimos tiempos registran mis estadísticas son los siguientes: 1. Dificultad de practicar la iridectomia periférica que se convierte en total, por exceso de dilatación pupilar, resultando que hemos practicado involuntariamente una extracción combinada. Rarísimo, si los enfermos están bien preparados. 2. Después de tallado el colgajo, algunas veces la pupila se contrae demasiado, dificultando el emplazamiento de la ventosa, lo que sin embargo se logra con un poco de habilidad. Caso de coger el iris entre la ventosa y el cristalino basta, después de haber dado la vuelta al cristalino dentro de la cámara anterior, interrumpir el paso del vacío, permitiendo la entrada en la ventosa de la presión atmosférica para que suelte la catarata, y cogerla nuevamente. 3. Rotura de la cápsula. (a) Esta puede ser instantánea al coger la catarata, lo que significa que nos hemos equivocado empleando un vacío demasiado intenso. Ocurre raramente. (b) En aquellos sujetos jóvenes con zónula muy resistente, puede abrirse el saco capsular por su borde inferior, y si bien es extraída la cápsula entera, su núcleo y gran cantidad de masas, quedan en las cámaras del ojo pequeños restos de substancia blanda. (c) Al acabarse la extracción, los labios del colgajo, pellizcando la catarata, pueden romper la cápsula; en este caso el contenido del saco capsular se derrama fuera de las cámaras del ojo, en los fondos de saco conjuntivales. Como se comprende, la rotura de la cápsula no tiene otro inconveniente que el convertir la extracción total en una quistiectomia. Es muy poco frecuente cuando se tiene práctica en el procedimiento. 4. Pérdida de vitreo; únicamente se presenta cuando el operador hace una presión indebida sobre el globo, o si el paciente contrae los músculos orbitarios. Rarísima, con la parálisis temporal del orbicular.

Las complicaciones post-operatorias, si la operación ha sido sin accidentes, y el enfermo no comete ninguna imprudencia durante los tres primeros días, no existen, salvo en rarísimas escepciones. Si hemos practicado la extracción combinada, es posible un enclavamiento en los labios de la herida de las fibras zonulares o de los ángulos del coloboma, ocasionándose retrasos en la cicatriz, iridociclitis plástica y glaucoma secundario. En los casos de extracción laboriosa, en los que se ha pellizcado el iris, puede sobrevenir ligera iritis. Si durante el transcurso de la operación se ha roto la cápsula, las complicaciones son análogas á las que se producen operando por quistiectomia. En los casos de hernia vitrea, si logramos seccionarla y

coaptar los labios de la herida con la sutura, queda la pupila negra, central redonda, y como única consecuencia ligeras opacidades del vitreo que desaparecen lentamente. En los enfermos indóciles, que se mueven, tienen tos, propensos al vómito o que reciben algun traumatismo, si la operación ha sido practicada con brecha periférica y sutura del colgajo, la complicación se limita, las mas de las veces, á una ligera reabertura subconjuntival de la herida y una gota de hifema. Complicación que desaparece con 24 horas de reposo sin ulteriores consecuencias.

Del estudio atento de las historias clinicas de mis operados se deduce que todos los accidentes operatorios y post-operatorios reconocen por origen una imprevisión, una falta de técnica, ó una indocilidad por parte del enfermo, y que el maximum de garantias consiste en la extracción simple con brecha periférica, sutura conjuntival previa y parálisis temporal del orbicular.

La falta de sintomas reaccionales debidos á la simplicidad de la maniobra quirúrgica, y á la ausencia de restos cristalinos en las cámaras del ojo, acortan la duración de la convalecencia y aseguran una agudeza visual normal.

OPERATION DE BARRAQUER

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L'extraction du cristallin dans sa capsule a été à différentes époques mise en pratique; ses avantages et ses inconvénients ont été souvent mis en discussion; mais en raison des difficultés inhérentes à son exécution sans que l'oeil ait à en souffrir, elle a été abandonnée et remise en honneur tour à tour. L'opération de Smith et celle de Barraquer viennent à nouveau de poser le problème de l'extraction totale.

J'ai publié la relation de 24 cas opérés par la méthode de Barraquer; de ces 24 cas, j'en avais opéré 6 avec succès sous la direction de Barraquer; il est inutile d'insister sur la sûreté que l'on possède quand on peut à chaque instant recourir aux conseils d'un maître. Lorsque on est livré à ses propres moyens on se trouve à tout moment devant une série de difficultés qu'il faut résoudre sur le champ.

Cette communication a surtout pour but d'exposer les accidents qui me sont survenus au cours de 46 opérations que j'ai pratiquées; leur description permettra à ceux qui voudraient essayer la méthode nouvelle de les éviter et de diminuer le nombre de leurs insuccès.

ERISIPHAQUE

L'opérateur doit porter une grande attention à la marche régulière de l'appareil. Lorsque on prend livraison de l'appareil, il est prudent de nettoyer d'abord le moteur, en y faisant passer un courant de pétrole; sinon la première partie d'huile qu'on y introduit revient chargée d'impuretés; on peut clarifier cette huile, en la filtrant sur du noir animal. Il est bon d'enlever l'huile après chaque séance d'opération, et de nettoyer ensuite le moteur au pétrole.

L'appareil porte-ventouse doit être d'une propreté extrême. On le nettoie à l'aide d'un petit bâton dont le bout taillé en pointe est recouvert de gaze. Il faudra passer un mandrin dans les divers trous et trajets, bouchés souvent par du sang ou des débris organiques entraînés par l'aspiration; il faudra nettoyer de même la ventouse.

Au début, pour m'assurer du fonctionnement, j'approchai la ventouse de l'oreille pour entendre le clapotement produit par la succion. Actuellement j'ai établi un système de contrôle qui me renseigne sur le champ sur le fonctionnement régulier de l'appareil. J'ai intercalé sur la trajet du tube d'aspiration un petit vacuomètre, à l'aide d'un tube en T. Lorsqu'on met le moteur en marche, on voit rapidement, au bout de 4 à 5 secondes, le vide atteindre 55 à 60 centimètres; puis je place la ventouse sur la peau du bras ou sur la lèvre, et j'établis la communication en pressant sur le bouton de l'appareil porte-ventouse; on voit d'abord une chute brusque du vide à 45 centimètres, puis une remonte rapide; on a à peine le temps de compter jusque 4 et déjà le vacuomètre est revenu à 60. L'appareil est alors en ordre; pour l'emploi il suffit de stériliser la ventouse dans la flamme d'une lampe à alcool. Je considère l'emploi du vacuomètre non seulement comme utile, mais comme indispensable; il permet de se rendre compte, à chaque instant, avant et pendant l'opération, de la marche régulière de l'aspirateur.

PREPARATION DE L'OPERE

Barraquer prépare le malade en vue de l'opération pendant plusieurs jours; après l'avoir couché il lui fait faire des mouvements des yeux. J'ai trouvé cette pratique inutile, je dirai même nuisible. J'ai constaté que les opérés que l'on traitait de cette façon pendant plusieurs jours, arrivaient à l'opération bien plus agités et anxieux que ceux que l'on se contentait simplement de coucher sur la table après leur avoir recommandé de se tenir tranquilles et de ne pas faire de mouvements brusques avec les yeux.

Tous les opérés sont examinés préalablement à la lampe à fente; on se rend ainsi compte beaucoup mieux de la forme de la cataracte; en même temps j'examine l'état de l'iris. C'est ainsi que sur 45 cas, j'ai trouvé 19 fois de la dégénérescence hyaline du bord pupillaire avec disparition plus ou moins étendue de la collerette; deux fois, il y avait en même temps de l'atrophie du feuillet postérieur de l'iris. La veille de l'opération, on lave abondamment les yeux, au sérum stérilisé; on s'assure de la perméabilité des voies lacrymales; on savonne le bord des paupières et on le badigeonne avec une solution de nitrate d'argent à 3%.

Une heure avant l'opération, on instille toutes les dix minutes un collyre à l'euphtalmine, à 4% contenant quelques gouttes de cocaïne à 5%, de manière à obtenir une dilatation pupillaire maxima qui est absolument indispensable. Puis on met de la cocaïne 5% dix minutes

avant l'opération. Je ne fais l'injection à la tempe pour la paralysie partielle des paupières que lorsque je me trouve en présence de malades indociles. Au moment d'opérer, un aide retourne la paupière supérieure, et la saisit entre les mors d'une pince, de manière à étaler largement le cul de sac conjonctival; l'aide écarte en même temps la paupière inférieure; une infirmière arrose alors abondamment l'oeil de manière à entraîner toutes les mucosités.

TECHNIQUE DE L'OPERATION

Le moteur est placé sur un plateau rond et calé à l'aide d'une vis; ce plateau est adapté à une bague mobile sur une tige verticale portée par un pied en fonte; la bague porte le vacuomètre et en même temps un petit plateau pour les instruments. L'appareil est placé à droite de l'opérateur. Avant de commencer, il est prudent de contrôler une dernière fois la marche de l'appareil; j'insiste sur le fait, parce qu'il m'est arrivé de devoir, après l'incision de la cornée, attendre pendant quelques minutes pour déboucher la ventouse. Pour écarter les paupières je me suis servi au début d'un écarteur de Desmares; actuellement j'emploie avec avantage un crochet à strabisme que l'on place et retire plus facilement.

L'aide écarte la paupière supérieure à l'aide du crochet à strabisme tenu d'une main; l'autre main écarte la paupière inférieure l'opérateur fait son incision cornéenne comme il a l'habitude de la faire; seulement le lambeau doit être plus grand, il doit occuper les $\frac{2}{5}$ de la cornée. L'incision est faite à l'union de la cornée et de la sclérotique, et terminée par un lambeau conjonctival.

L'incision faite, l'on retire le crochet à strabisme, et je pratique alors une petite iridectomie soit totale soit périphérique à la manière de Hess. Je maintiens moi-même la paupière supérieure en la pressant contre le rebord orbitaire à l'aide du petit doigt de la main gauche qui manie la pince ou le crochet de Tyrrel.

S'il y a du sang dans la chambre antérieure, je lave abondamment avec du sérum stérilisé. Ensuite je replace moi-même le crochet à strabisme de la main gauche et je relève la paupière tout en la tirant en haut. Je ne commande pas au malade de regarder, en bas je lui demande de se tenir tranquille. A ce moment le moteur est mis en marche; quand le vide atteint le degré voulu de 55 à 65 centimètres, j'introduis latéralement la ventouse, en dehors pour l'oeil droit, en dedans pour l'oeil gauche. La ventouse passe sous le bord pupillaire. Après l'avoir délicatement posée sur le cristallin, on presse le bouton

pour établir la communication entre la ventouse et l'aspirateur. A ce moment, le vacuomètre descend vers 45c. pour remonter immédiatement à son point de départ. Alors on peut retirer la ventouse en faisant glisser le cristallin sur la fossette patellaire, tout en faisant basculer le manche de l'instrument en avant. Ou bien on fait exécuter au cristallin un cumulet de manière à ramener sa face postérieure en avant; il faut pour cela faire basculer le manche en arrière.

Le cristallin sorti, on instille de l'ésérine huileuse; on attend quelques minutes avant de faire la réduction de l'iris; j'emploie pour cette manoeuvre une spatule coudée à angle droit.

Comme pansement, je place le binocle avec une bande, ou bien, comme Barraquer, je maintiens les bourdonnets d'ouate à l'aide de bandelettes croisées. Si l'opéré ne se plaint pas, j'enlève le pansement de l'oeil non opéré le 4ième jour, celui de l'oeil opéré au bout de 7 à 8 jours. Le pansement reste donc en place plus longtemps que dans l'opération ordinaire. J'ai vu une hernie du vitré se produire chez une malade privée de pansement le 6ième jour.

Telle est la marche d'une opération normalement conduite.

ACCIDENTS OPÉRATOIRES

Une plaie trop petite empêche l'introduction facile de la ventouse, et surtout arrête la sortie du cristallin. On doit agrandir la plaie, en sectionnant un des angles avec des ciseaux droits à branches courtes et fortes.

Si le lambeau conjonctival commence trop bas, il empêche l'introduction de la ventouse; cet accident m'est arrivé deux fois. Pour éviter cet inconvénient, il faut changer le point d'introduction, ou même faire soulever le lambeau conjonctival à l'aide d'une pince.

Le pincement de l'iris provoque de la douleur, et aussi de l'hémorragie; on peut même en retirant la ventouse, produire un décollement de l'iris. En cas de pincement, il faut lâcher le bouton de pression et réintroduire la ventouse.

L'hémorragie dans la chambre antérieure disparaît toujours facilement par lavage; dans un cas cependant, il s'était formé un grand caillot, que je ne parvins pas à enlever. Je me suis décidé à transformer l'opération de Barraquer, en une opération de Smith; en pressant sur la partie inférieure de la cornée à l'aide d'un crochet à strabisme, j'ai pu faire sortir le cristallin sans perte de vitré; la vision de cet opéré fut de 0.1 avec + 11 dioptries.

L'accident le plus fréquent est celui qui provient d'un défaut dans

l'adhérence du cristallin à la ventouse; la ventouse lâche le cristallin, soit au moment où on veut faire l'extraction, soit lorsqu'il se présente déjà dans la plaie: un bruit de friture annonce l'accident. Il faut alors retirer rapidement la ventouse. Dans un cas, le cristallin s'est engagé dans le trou d'une iridectomie périphérique; avant de réintroduire la ventouse, j'ai sectionné le bord pupillaire qui avait été conservé. Si les circonstances restent favorables, on peut réintroduire la ventouse plusieurs fois; il m'est arrivé de réussir à la 4^{ième} reprise; mais il faut naturellement dans les tentatives répétées, veiller à ne pas aspirer le vitré. Le cas suivant extraordinaire, mérite d'être rapporté. Je faisais une troisième tentative d'aspiration chez un de mes opérés, quand tout d'un coup j'entends un bruit de succion, je vois l'oeil s'affaisser complètement; malgré l'énorme perte de vitré je me décide sur le champ à faire l'extraction du cristallin à l'aide de l'anse de Snellen; la manoeuvre réussit mais l'oeil est tout ratatiné; la cornée déborde la lèvre scléroticale, en outre il s'est formé dans la sclérotique un pli que je m'efforce de réduire. Je m'attendais naturellement à un désastre. Les jours suivants le malade ne se plaint pas; j'enlève le pansement le 7^{ième} jour. A mon plus grand étonnement, l'oeil s'est reconstitué complètement; la plaie est fermée; le malade voit. Actuellement la vision est de 0.6 avec 13 dipotries. De toutes les opérations que j'ai faites, c'est la plus extraordinaire.

Un autre accident consiste dans la rupture de la capsule, trop faible pour résister à la traction exercée par le vide; on se trouve alors en présence d'une extraction avec discission que l'on termine suivant les règles ordinaires.

La luxation du cristallin constitue l'accident le plus grave. Parfois le cristallin est luxé directement en haut; on peut le faire glisser en bas à l'aide d'une spatule et puis tenter de nouveau l'aspiration. Ou bien il bascule en arrière, le vitré venant se placer au devant; dans ce cas il ne reste que la seule ressource de recourir à l'extraction à l'aide de l'anse de Snellen ou de Taylor. On l'introduit rapidement derrière le cristallin, puis on reporte le manche en arrière; au lieu de retirer le cristallin en l'appliquant contre la cornée, on le fait glisser sur l'anse à l'aide d'un crochet à strabisme appliqué extérieurement sur la cornée. Cette manoeuvre est moins brutale et expose à moins de perte de vitré.

L'écoulement de vitré avant l'introduction de la ventouse ne m'est pas arrivé. Après la première introduction, le corps vitré peut se

placer au devant du cristallin, sans qu'il y ait luxation; dans ce cas il faut faire l'extraction par discission. A deux reprises, il m'est arrivé le singulier accident suivant: Après une première tentative inutile, l'iris prend la forme d'entonnoir au fond duquel se trouve le cristallin. La cornée bombée, rigide au lieu d'être affaissée, est séparée du cristallin par un grand espace; quand on introduit une spatule, la cornée s'affaisse.

L'écoulement du vitré peut se produire après la sortie du cristallin. Parfois après la sortie, le vitré se présente un moment dans la plaie, puis rentre aussitôt. Parfois au lieu de s'écouler, il vient former une boule entre les lèvres de la plaie: il vaut mieux dans ce cas exciser la hernie du vitré avec les pince-ciseaux. Si au bout de quelques jours la hernie persiste, je n'hésite pas à la recouvrir à l'aide d'un lambeau conjonctival; dans deux cas traités de la sorte, j'ai obtenu une bonne et rapide fermeture de la plaie.

SUITES DE L'OPERATION

Il y a souvent de la kératite striée qui disparaît assez rapidement sans laisser de traces. Dans un cas de kératite striée, le corps vitré était venu s'accoler à la face postérieure de la cornée et y déterminer une forte opacité.

L'enclavement de l'iris dans l'un des angles de la plaie s'est produit dans une demi douzaine de cas. Dans 7 cas il y a eu une déformation de la pupille déviée vers le haut. Cette complication est moins grave que dans l'extraction avec discission. Dans un cas j'ai eu après une extraction normale, une hernie du vitré constatée le 6 ième jour.

Il est intéressant d'examiner au bout de quelque temps les opérés à la lampe à fente. On constate dans le champ pupillaire que le vitré tombe en avant; dans un cas opéré sans iridectomie, j'ai trouvé le vitré sous forme de champignon pédiculé, faisant saillie à travers l'ouverture pupillaire. J'ai obtenu un décollement de la rétine, consécutif à l'opération.

Barraquer a écrit qu'il ne pouvait se former d'adhérence entre l'iris et le vitré; j'ai vu un cas où il s'était produit une adhérence très nette entre le vitré et le bord pupillaire; l'extraction s'était faite normalement.

Dans deux cas où j'ai dû employer l'anse, il s'est formé aux dépens de masses cristallines, un amas de substance blanche, masquant complètement la pupille; dans un cas la résorption est presque complète; dans le second la résorption est en train de se faire.

Si je résume les cas voici les visions obtenues:

Vision 0 dans deux cas d'insuccès complet.

un cas de choroïdite maculaire. (Résultat opératoire parfait.)

2 cas de chorioretinite spécifique ancienne (idem).

1 cas de décollement de la rétine antérieur (idem).

Vision 0.1 6 cas.

0.2 7 cas.

0.3 6 cas.

0.4 5 cas.

0.5 1 cas.

0.6 4 cas.

0.7 4 cas.

Vision 0.1 à 3 mètres 1 cas.

0.2 à 2 mètres 1 cas.

0.1 à 1 mètre 1 cas.

Le point le plus délicat de l'opération de Barraquer, celui dont dépend le succès, est le moment de la pression sur le bouton de l'appareil porte-ventouse; il suffit d'un instant d'inattention, lorsqu'on fait la traction pour la manoeuvre de sortie, pour que le vide diminue, et que le cristallin retombe. C'est pour moi le défaut de l'appareil; le vide ne devrait pas dépendre d'un mouvement involontaire; il devrait rester permanent et ne pourrait disparaître que par une manoeuvre volontaire. C'est pour remédier à cet inconvénient que nous avons essayé de rendre le système d'occlusion indépendant de la ventouse. A cet effet, la ventouse est placée à l'extrémité d'un manche long de 7 centimètres et de 7 mill, de diamètre; l'appareil porte-ventouse contenant les soupapes est intercalé dans le trajet du tuyau en caoutchouc entre le vacuo mètre et la ventouse. La disposition est représentée dans la photographie ci-contre (Fig. 1). La ventouse se manie comme le manche d'un instrument ordinaire. Un aide presse sur le bouton au commandement de l'opérateur, quand la ventouse est en place; l'aide signale le moment où le vide a de nouveau atteint le degré voulu; la manoeuvre d'extraction commence alors avec plus de sûreté et de chance de succès que lorsqu'on se sert du dispositif de Barraquer.

Il faudrait probablement modifier la ventouse aussi, de manière à rendre plus intime l'adhérence du cristallin. Quand cette dernière condition sera réalisée, je crois que les chances d'insuccès seront réduites au minimum.

Y A-T-IL UN VIDE VIBRATOIRE?

L'existence du vide vibratoire produit par l'érisiphaque a été mise en doute; nous avons le docteur Kleefeld, et moi, essayé de le mettre clairement en évidence et même de l'inscrire. Kleefeld a d'abord noté des vibrations en introduisant le tuyau aspirateur de l'érisiphaque dans l'entonnoir d'un appareil de physique appelé flamme sensible. Ensuite nous avons eu recours à un dispositif imaginé par le pro-



Fig. 1

fesseur de physique Henriot. Le dispositif employé consiste en une capsule manométrique, formée d'une boîte cylindrique dont une des bases est plus dépressible que l'autre. Deux pertuis y sont ménagés, l'un pour faire l'aspiration, l'autre pour être mis en rapport avec l'appareil porte-ventouse. Le contrôle du vide se fait à l'aide du vacuomètre. Un fil métallique est tendu à la face extérieure de la capsule, et rattaché aux deux extrémités du diamètre vertical; ce fil soutient

une lamelle métallique qui s'appuie sur la surface de la capsule, il porte un petit miroir concave. En face de la capsule on place comme source lumineuse une ampoule électrique, type monowatt. L'image de cette lampe est concentrée sur une règle graduée en celuloïde; on peut ainsi suivre le mouvement de la mouche lumineuse le long de la graduation. Au moment où le vide s'établit, on voit la mouche se déplacer rapidement sur l'échelle, en même temps le filament devient flou.; il en est de même lorsque on fait manoeuvrer les soupapes de l'appareil porte-ventouse. Si on remet le filament au point par interposition d'une lentille convergente entre le miroir et l'échelle, on constate la netteté absolue du filament quand le vide maximum a été atteint. Cet appareil expérimental tout en réunissant certaines conditions de sensibilité ne nous a pas permis d'inscrire des vibrations. La déformation de la paroi de la capsule sous l'action de la pression atmosphérique diminue les possibilités d'une transmission de vibrations existant à l'intérieur du système d'aspiration.

Pour arriver à se rendre compte des mouvements de l'air dans le tuyau d'aspiration, Kleefeld a imaginé le dispositif suivant: dans un tube de verre d'un calibre à peine supérieur à la section intérieure de la tuyauterie de caoutchouc, on suspend une feuille d'or. La minceur, l'extrême malléabilité, l'absence de vibrations propres, le poids négligeable, font de la feuille d'or un indicateur précieux des mou-



Fig. 2

dres vibrations d'un système gazeux. On interpose cet indicateur entre l'érisiphaque et le porte-ventouse. On place le système dans une lanterne à projection. L'ombre de la feuille d'or est recueillie sur un écran, ou bien inscrite sur une plaque photographique. Quand on met l'érisiphaque en marche, on voit sur l'écran, la feuille d'or animée d'un mouvement de vibration très intense, tandis qu'elle se soulève assez rapidement dans la direction de l'aspiration. Au bout d'un certain temps cette vibration paraît cesser et la feuille d'or ne présente plus que quelques soubresauts; elle conserve cependant l'inclinaison qu'elle avait acquise au début de l'aspiration.

Pour inscrire les vibrations, nous remplaçons l'écran par un châssis

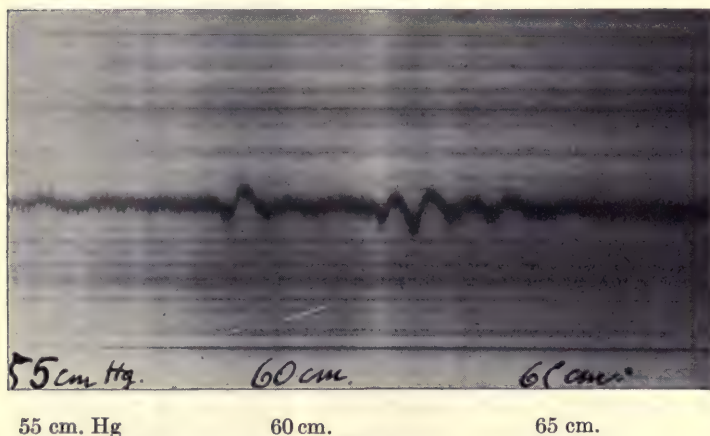


Fig. 3

dans lequel on fait glisser une plaque photographique; la plaque passe en un mouvement régulier et continu, devant une fente horizontale. Nous avons ainsi obtenu les tracés ci-joints (Figs. 2 et 3), qui montrent nettement les oscillations. Nous avons ainsi la preuve que l'érisiphaque de Barraquer produit un vide oscillant ou vibratoire.

Si on peut affirmer que ce vide vibratoire existe, il est plus difficile de dire quelle est sa part d'intervention dans le mécanisme de l'extraction. A-t-il une action prépondérante?¹ On pourrait peut-être augmenter l'effet des vibrations en imprimant à un moment donné, des

¹ Peut être l'aspiration agit-elle en déformant la surface du sac cristallin. La capsule du cristallin est attirée dans la ventouse; en même temps elle exerce une traction sur les fibres de la zonule qui se tendent et finissent par se rompre. L'action des oscillations serait ainsi renforcée.

oscillations directement à la ventouse et favoriser les chances de rupture de la zonule.

Quoiqu'il en soit l'érisiphaque de Barraquer est un instrument qui est venu donner à l'extraction totale de la cataracte, une vogue nouvelle; il supprime la pression sur le corps vitré qui est si brutale dans l'opération de Smith; sous ce rapport, l'opération de Barraquer sur celle de Smith; Si on a des déboires dans un certain nombre de cas, déboires inévitables dans les premiers essais, du moins dans ceux qui se terminent avec succès, le résultat donne de grandes satisfactions; pas d'irritation, une pupille noire, une vision excellente, et surtout pas de cataracte secondaire à redouter. Ce sont des avantages dont il ne faut pas méconnaître le prix.

Avant de pouvoir se prononcer sur la valeur définitive du procédé, il faudra que la méthode soit largement mise en pratique. Il en a été de même de l'extraction avec dissection. Que l'on relise le nombre incalculable de travaux publiés sur ce sujet, les discussions dans les congrès, on verra qu'il a fallu des années pour établir les règles qui nous ont amenés à la précision obtenue par le procédé opératoire de la dissection. Il en sera de même pour l'extraction totale par l'érisiphaque ou pour toute autre méthode semblable. Il faut que chacun aborde cette étude sans parti pris; en y apportant la sincérité que l'on doit exiger dans toute recherche scientifique. Pour moi, malgré les insuccès que j'ai signalés, je reste convaincu que l'opération de Barraquer est appelée à un grand avenir et qu'elle ne tardera pas à entrer dans la pratique courante.

DISCUSSION OF PAPERS OF PROFESSORS BARRAQUER AND GALLEMAERTS

DR. LUCIEN HOWE (Buffalo, N. Y.): Although I have not seen this operation, the principle described is not new. As I understand it, some suction must in reality be made, otherwise there would be no need of what is practically a suction pump. Many years ago, when Knapp reported a series of extractions, he said, in substance, that while the "vis à tergo" principle served our purpose, it would be much better if we could have some way of drawing out the lens by a "vis à fronte." It then occurred naturally to me, as probably to many others, to employ some arrangement by which suction could be made. I tried, but failed at first, because the suction sucked out lens and vitreous also. Therefore I simply attached a small rubber bulb to a short tube with a minute elliptical opening, as shown in the accompanying illustration. That serves the purpose admirably. But not being one of ardent advocates of extraction in the capsule, this suction bulb is used only in exceptional cases. When, however, we reach the embarrassing point where

the lens appears and we cannot make it come out properly, it is of real advantage to employ slight suction, applying the tube to the edge of the lens in such a way that it can be tipped from side to side.



I think the older men are rather inclined to follow the advice of one of our American poets, who was also a physician, and say with Holmes:

"Be not the first by whom the new is tried
Nor yet the last to lay the old aside."

DR. JOHN WESTLEY WRIGHT (Columbus, Ohio): My conception of the intracapsular operation for cataract was the result of fortuitous circumstances in my early experience in the flap and combined methods prevalent at that time, which because of an extensive incision of one-half of the corneal circumference and an equally extensive flap, resulted too frequently in imperfect coaptation, a prolapse

of the iris, a drawn-up or obliterated pupil, and more or less corneal opacity. To avoid such discouraging conditions I conceived an incision much smaller, less liable to gap, and of such dimensions as to permit the ready delivery of the lens.

Briefly, the incision is made entirely within the cornea, the puncture and counterpuncture being one-third down the corneal circumference, at the sclerocorneal junction, and completed at a point 2 mm. within its upper border. With this incision I discovered that pressure upon its upper segment, in addition to making the opening for the delivery of the lens much larger than in the usual methods, caused a detachment of the lens from the annular ligament at its upper portion, when with careful pressure it is stripped out from the surrounding attachments and its delivery in capsule was readily affected. Pressure thus made causes a displacement of the vitreous in such manner that there is a tendency for it to seek a point of least resistance, thus forcing the lens toward the open gap caused by the incision.

After considerable experience with the technique and realizing its importance, a description of the operation was published in the Columbus Medical Journal, October, 1884. Since its conception I have practised this technique with general satisfaction during my professional career.

The pupil, as a rule, dilates widely under the effects of a mydriatic and no injury to the iris results. For this reason I rarely find it necessary to make an iridectomy. The lens, therefore, with gentle pressure, will pass through the pupillary space without bruising or lacerating the pupillary border. I have knowledge of cases of dislocated lens where, without any effort whatever on the part of the patient, it passed readily from the posterior chamber to that of the anterior, and back again in the absence of the effects of a mydriatic.

I have the history of a case where the transition was readily effected at the pleasure of the patient by certain movements of his head.

If the pupil does not dilate well, it is useless to attempt to force the lens through. This applies whether within its capsule or in capsulotomy. Then I make a small iridectomy, or otherwise an iridotomy, by slitting the iris slightly from the center of its superior border toward its periphery. My experience is that a small iridectomy or an iridotomy, as indicated, will facilitate the passage of the lens as efficiently as a large one.

The posterior surface of the lens lying as it does against the vitreous, it is not unusual for adhesions to occur between their supporting membranes; this, often at the hyaloid fossa, although they may occur at other points, when the attempted removal of the lens in capsule is liable to rupture the hyaloid membrane, with more or less loss of vitreous, or, on the other hand, should a rupture of the lens-capsule occur, fragments of it will remain adherent to the hyaloid membrane, and interfere with vision to a greater or less extent. In such cases capsulotomy has no advantage over the intracapsular, inasmuch as the remaining capsule interferes with the visual acuity in either method. These are conditions that cannot be determined prior to operation.

There are cases where the lens cannot be delivered within its capsule without the risk of serious injury or loss of the eye, generally for the reason that it is so firmly attached to its supports. This condition cannot be definitely diagnosed before an attempt to dislocate has been made. My experience has taught me about how much pressure should be made with safety, and I do not usually go beyond that point. If the lens does not readily become detached from its moorings, then I do capsulotomy, because in this event I consider it the safer operation. I have succeeded in dislocating the lens in some instances without difficulty where there had been considerable iritic adhesions.

The point upon which pressure is made is an important matter. It should be made upon the center of the upper segment of the cornea, near the incision, and the counterpressure, simply enough to steady the eye, on the opposite side of the cornea. When applied near its center, it causes contraction of the opening and impedes the delivery of the lens. Besides, it frequently causes buckling or kinking and occasionally leaves slight opaque streaks in the cornea. This is entirely avoided with the pressure above the incision.

DR. JOHN O. McREYNOLDS (Dallas, Texas): I am fully conscious of my inability to present a comprehensive analysis of the paper of Prof. Barraquer, and I can only hope to offer a few conclusions from my observation of his work in Barcelona, in New York, Boston, Philadelphia and Richmond, together with some personal experience with his method during the past year.

The immediate results and the impressions produced upon the profession have varied exceedingly in different cities. In Barcelona, and indeed throughout Spain, the procedure has met with a most enthusiastic endorsement. In New York and Boston the results were not up to the standard, while in Philadelphia and Richmond the operations were performed with practically uniform success. In Richmond, 14 operations were made in one day, and in only one case was there any rupture of the capsule with a tiny bead of vitreous

lost. In Philadelphia there were 9 extractions, with no vitreous lost, in the Wills Hospital, the Jefferson Hospital, and the Polyclinic Hospital.

In Boston there were 5 operations made at the Massachusetts Eye Infirmary on the first day, with 3 cases of ruptured capsule, in two of which there was also some loss of vitreous. The rupture of the capsules in these cases was evidently due in large measure to the fact that the current was much too strong. This difficulty was remedied on the second day, when 2 extractions were made without accident of any kind.

In New York, 2 extractions were made at the Knapp Memorial Hospital, without accident; 1 at the Manhattan Eye Hospital, without accident; 3 at McLean's Hospital, 2 of which were correct, and one had expulsive hemorrhage immediately following the incision before any effort at extraction was made. Seven operations were made at the New York Eye & Ear Infirmary, with 3 capsules ruptured, including 2 cases of vitreous loss.

In the four American cities there were 43 extractions with 5 cases of slight vitreous loss and 6 cases of ruptured capsule, while in the last 23 cases there was only one case of a tiny bead of vitreous loss. In the last series of 23 cases Prof. Barraquer had the same assistant, Dr. Poyales, of Madrid, who employed regularly lid retraction instead of the speculum, and the result was approximately 4 per cent. of vitreous loss.

Conclusions.—1. In the hands of an experienced and skilful operator the method can be developed to a point where vitreous loss will become an unimportant factor.

2. An uncomplicated rupture of the capsule reduces the operation essentially to a capsulotomy operation.

3. There is no increase in the hazard of choroidal hemorrhage.

4. With a peripheral buttonhole iridectomy, which is regularly employed when possible, there should remain a permanently mobile pupil without any incarceration of the iris.

5. With a conjunctival flap adequately held in position with sutures, according to Prof. Barraquer's method, the healing process should be sufficiently complete after one week to permit the patient to leave the hospital, provided reasonable supervision is maintained.

6. The danger of infection and postoperative inflammation of all kinds should be reduced to the minimum.

7. Much can be accomplished by a critical study of the intimate structure of each lens to be operated, so that the form of operation may be modified to meet the requirements of each individual case.

8. The method should not be undertaken without previous abundant experience with the older and more established measures of dealing with all forms of cataract, because it is more difficult of execution and developments may occur which only experienced hands can safely control.

9. It has a definite place in ophthalmic surgery as the ideal to be attained through unremitting diligence, experience and care.

DR. LOUIS D. GREEN (San Francisco, Cal.): Dr. Barraquer deserves great credit for reviving the suction operation by putting it on a firm basis, thereby placing the intracapsular operation many steps forward. Ophthalmologists

who have mastered the intracapsular operation and performed it consistently by one of the two or three methods over a period of years, commend it highly. But even for the most skilful, there was much that was left to be desired. By the forceps method, too many capsules were ruptured so that one succeeded in doing the intracapsular operation in only a small proportion of cases. The Smith-Indian method requires a high degree of skill, only acquired after a great deal of practice on a large number of cases. The Barraquer method requires highly developed apparatus and skill in its use, and even then the considerable traction necessary in certain cases with tough suspensory ligaments may result in a detachment of the choroid or retina; or if the capsule breaks, vitreous is liable to be drawn into the tube.

It is only natural that every surgeon should prefer to use the instruments with which he obtains the best results, and in the hands of Dr. Barraquer his apparatus undoubtedly does all that one could desire. In our hands, however, we find it has certain mechanical inconveniences that interfere somewhat with the delicacy of manipulation. We have accordingly modified it so that the valve is controlled by the foot, permitting the use of a very light cannula that is held in the hand, as delicately as a cataract knife, thus relieving the fingers of any cramped position.

According to the manner in which cataracts are removed, operations may be classified as extractions or expressions. The classical capsulotomy and the Smith-Indian operation are expression operations. The Hulen or Barraquer vacuum and Stanculeanu or Knapp forceps methods are extraction operations. We have gradually evolved a technique which is a combination of expression and extraction, and from an experience of over 100 cases that we have operated by this procedure, we believe it minimizes, to a great extent, the dangers in either the Smith-Indian operation or the vacuum operation as advocated by Dr. Barraquer. This we have described elsewhere.

Briefly, the method consists of making a full half section of the cornea with a conjunctival flap. The flap is grasped and cornea raised with a special delivery forceps which also act as a delivery hook, and are held in the left hand, and the cannula held in the right hand is introduced and lightly placed upon the cataract. The foot valve is now engaged and a few seconds allowed for the spoon to become firmly attached to the anterior capsule and lens; the cannula is gently raised so that the upper border of the cataract is tilted forward into the wound, at the same time drawing the cataract out of the eye. At the moment when rotation for the delivery of the lens starts, the left hand releases the conjunctiva and the forceps which held it are shifted to the lower part of the eye near the limbus, and by gentle pressure assists in delivering the lens. We thus attain an expression-extraction with a division of the forces in each and with less trauma to the eye than would occur if either procedure were used alone.

COL. R. H. ELLIOT (London, England): I had not wished to speak to-day, but several of my American friends have asked me to do so on the strength of my having had some cataract practice in the East. I am conscious on this and similar occasions of the warning uttered by Gamaliel before the San-

hedrin, if I may change one word, "Lest by any means we may be found to fight against the truth."

I want first of all to differ with Dr. Barraquer most respectfully in the analogy he has more than once used between the capsule laceration operation and the removal of cysts from the abdomen. I respectfully submit, that there is no such analogy. The irritating contents of such cysts and the nature of their walls, and the fact that they will fill or grow again if not completely removed, put them in an entirely different class from the lens inside its capsule. I emphasize this because many of us with large experience in cataract and the capsule laceration operation can confidently rely upon getting from 90 per cent. to 96 per cent. of good results from that operation.

But the side of the question that I want to bring before you today—please understand that I am not fighting against this operation, but I do want you to think squarely about it—is related to what is often called the "diaphragm of the eye," that is, the ciliary body with the suspensory ligaments and capsule. This diaphragm is a great protection to the eye, both from the mechanical escape of vitreous, and also from infection. It powerfully strengthens the anterior hyaloid membrane. That is a matter on which any man with large cataract experience can have no question in his mind. I look upon—and always will look upon—the integrity of the vitreous at the close of a cataract operation as a very great asset. It is not merely that in these cases you are apt to get an escape of vitreous, but that both in the Smith operation and I believe also in Dr. Barraquer's operation, there is a tendency to a damage of the upper part of the hyaloid body leading to a definite tendency to herniation of that part. That was observed by Col. Kirkpatrick working with the Smith operation in Madras. It has been observed by Dr. Wright working with the Barraquer operation. Within the last few days an exponent of the Barraquer operation with whom I raised the question was good enough to tell me that he had observed such a tendency to herniation with the Gullstrand slit lamp. I do not think you can look upon any operation that weakens the upper part of the hyaloid and tends to herniation, and to impaction of that body in your wound, without some apprehension.

I hold that the sound thing for the younger operators to do is to first of all select a safe operation. Then when they have acquired manipulation facility, when they have learned all that the old operation has to teach, they can make up their minds for themselves whether they ought or ought not to undertake such a procedure as this. I can only say, that if in the course of years I have a cataract to be removed, I shall go to a surgeon who will do a preliminary iridectomy, who will lacerate the capsule, and who will subsequently do a discission. I admit you can get very beautiful results with the Barraquer operation, but if today you were having the two—the old and the new procedures—presented to you for the first time, with all that can be said for and against both of them, I think you would pause before you took up the intracapsular operation. Judge the method logically, look at it in the large, and make up your mind. If you are going to do it, watch your records very carefully and do not forget to look for impaction of the hyaloid and for disturbance of the transparency of the hyaloid body.

PROF. F. DE LAPERSONNE (Paris, France): En attendant que le traitement médical, que les sérums et vaccins nous permettent de prévenir ou de guérir la cataracte, l'idéal opératoire est de faire l'extraction dans la capsule. Le procédé de M. Barraquer marque un grand progrès, mais ce n'est pas une opération de tout repos. Les perfectionnements successifs et les si justes observations de mon ami le Professeur Gallemaerts, en sont les meilleures preuves.

Trois facteurs principaux interviennent (1) l'opérateur; tout le monde n'a pas la grande habilité de M. Barraquer pour cette opération: je ne conseillerais jamais à un jeune oculiste de faire sa première cataracte par ce procédé: même pour un oculiste exercé, il faut un stage, qui se fait aux dépens du malade; en plus le diagnostic des indications opératoires, le calcul exact, fait à l'avance, de la résistance de la zonule me paraissent très difficiles. (2) il faut avoir un aide très sûr pour éviter toute pression (3) il faut enfin que l'instrument soit encore perfectionné.

DR. LLOYD MILLS (Los Angeles, Cal.): When Prof. Barraquer published his first paper in the *Siglo Medico*, I read his work with the conviction that it promised to be an improvement upon the Smith intracapsular operation which I was then using in selected cases with normally variable success. My attempts to get in touch with Prof. Barraquer failed, and, without knowing the detail of his instrumentarium, I took up the problem by attempting the vacuum extraction of kittens' lenses, using an aluminum-bodied erisphake with an air vent in the side. This vent was so much larger than the tube which carried air to the suction cup, that the cup could be applied to the lens with the suction already active and yet without engagement between cup and lens, until the side vent was closed by my finger, when the lens was drawn at once into the cup. As a source of negative pressure I used successively water suction, the electric vacuum apparatus used for ridding the throat of blood in tonsil work, and finally connected our operating room directly with the great steam vacuum exhaust of the engine-room, capable of developing a vacuum of 18 inches, which, of course, was far in excess of any practical needs.

The first lesson learned was that the lens slipped away from the cup with too little vacuum; the second was that too sharp suction ruptured the capsule; the third was that with strong suction applied directly to the central part of the lens and using a pull straight forward the lens could be extracted in capsule, but practically always with the entire vitreous body attached. Only by rocking the lens with some vigor, not radially, as in turning a wheel, but in the anteroposterior plane, could an uncomplicated delivery of a lens in its capsule be obtained. A study of this method led me to believe that the delivery of lens, with vitreous attached, came from the lack of entrance of aqueous or air into the patellar fossa, for, when this was accomplished by the simple gentle insertion of a narrow grooved spatula behind the lens without rupturing the hyaloid, and with atmospheric pressure behind the lens equal to that in front of it, normal intracapsular extraction became the rule and not the rarity. May I suggest that possibly the entrance of fluid or air

into this situation is an additional and important accomplishment of vibration?

In the light of our constant experiences with the rise and fall of surgical innovations in general I feel that any criticism, and especially that any exaggerated approval of Barraquer's method, should be withheld until it has been tested analytically by competent men and along the exact lines which the master himself has so painstakingly worked out over so many years. It seems to me to be the height of presumption, and our medical editors are partly responsible for this, that men should be permitted to rush into print after a few performances of a surgical novelty and then advise extreme modifications of a method whose exacting details as a rule they have never fundamentally acquired. We saw the evils of this on the Pacific coast in the propaganda for another form of cataract operation now quietly sinking to its natural surgical level, when the entire profession of that coast, and indirectly many of our patients, were so circularized as to the easy adaptability of this method to all kinds and conditions of cataract that those who did not acquire this technique were at times stigmatized as laggards, if not actually considered to be to some extent incompetent. The constancy with which any surgical method gives success to the average qualified operator is its real measure of merit, and it is sincerely to be hoped that the development of Prof. Barraquer's brilliant operation in America, if it is to develop here, will be done in that orderly and decent fashion which the scientific attitude of mind properly demands.

DR. JOS. A. WHITE (Richmond, Va.): In a rather long professional career I have had some little experience with cataract extraction, and have become wedded to a capsulotomy operation with an iridectomy, preferably preliminary, if I can get my patient's consent. This has been reasonably successful with me in not a large series of cataract extractions, but something over 1500, which is enough to judge of results, and I believe that it offers more chances of restoring sight than any of the more recently suggested operations. There is no question as to the fact that the intracapsular method has come to stay, but I have been loath to attempt it. The first time that I saw the Smith-Indian operation there were two cases. The pressure applied was so great that expression of the lens was followed by considerable loss of vitreous, which is not to be regarded lightly. This operation, however, is reported as very successful in the hands of those who have had a large experience with it. Many modifications of the intracapsular operation have been suggested to diminish pressure and lessen traumatism, and as they come more to what I consider the safety line it is possible that I may undertake the procedure.

Recently I saw Prof. Barraquer do his operation under favorable circumstances. I was fortunate, with the help of my colleagues, to offer him 18 cases of cataract. Owing to a little secretion in the eye of two cases, a slight dislocation of the lens in one, and not sufficient light projection in the field of another, Prof. Barraquer rejected four and operated on the balance. Of these operations he had two ruptured capsules, but he removed the capsule apparently completely. There was only one case where a little vitreous presented at the end of the operation. Later I returned to the hospital, and none

of the patients had the slightest discomfort from the time of the operation, except one, and all seemed to be doing well. Notwithstanding the brilliant operations that I saw done by the intracapsular methods, if I had a cataract I would have a preliminary iridectomy done, the lens extracted after capsulotomy, and take the chance of capsular or after-cataract. I am satisfied that the old operation will hold its own among us for the reason that the intracapsular method is not suited to certain forms and to certain ages.

MR. J. GRAY CLEGG (Manchester, England): I have never done the operation, but I have seen friends of mine do it in London. When all is well, undoubtedly it is an ideal operation. What I am particularly interested in is the after-results, and I wish therefore to ask Prof. Barraquer as to the length of time of recovery and what post-operative complications occur; whether he often finds detachment of the choroid and other intra-ocular conditions which detract from the final results. These, it appears to me, are points of great interest, quite apart from the question of the operation itself.

DR. S. GEMBLATH (Paris, France): Dans une opération de cataracte, le but primordial, est l'amélioration de la vue du malade. Monsieur Barraquer ne nous donne aucun détail sur le résultat fonctionnel de l'oeil opéré. A cause de la manoeuvre opératoire assez traumatisante, on peut craindre des iridocyclites traumatiques plus ou moins tardives.

D'autre part, la large incision cornéenne nécessaire pour permettre la sortie à la fois du cristallin et de la canule, doit nécessairement, il me semble, donner lieu à un astigmatisme plus fort que celui des méthodes usuelles.

DR. W. A. FISHER (Chicago, Ill.): The first paragraph of Prof. Barraquer's paper states that the simple total extraction is the only rational method of operating for cataract, and his conclusions state among other things that loss of vitreous is rare with paralysis of the orbicular, and that it occurs only when the operator uses undue pressure on the globe or the patient contracts his orbital muscle.

If his conclusion is well founded, and I believe it is, then the first paragraph of his paper must be true—that the simple total extraction is the only rational method of operating for cataract. I believe Prof. Barraquer has given us a method of removing senile cataracts that has come to stay.

In his hands it appears to be perfect, but I do not believe experienced operators will encounter more difficulties by this method than by other methods. Prof. Barraquer's personal skill makes his operation appear quite simple, but such is not the case, and the same can be said of all methods.

Recently I had the pleasure of seeing Prof. Barraquer operate upon nine cases in Philadelphia, and fourteen in Richmond, Virginia. There was not a trace of vitreous loss in Philadelphia, and in only one case was there a loss of vitreous in Richmond, and in that one only a drop. But I do not believe his operation will supplant all other methods. Twenty-three operations performed among strange surroundings by any method with only one vitreous loss and that one only a drop, with a prospect of little if any post-operative inflammation, no after-cataracts and good vision in all of them, with the pupils all round and looking normal, is more than most operators

expect. Prof. Barraquer has achieved the above results in twenty-four consecutive operations, and I feel, as I believe most of you do, that he should be congratulated and encouraged, and I am sure the profession will be benefited by his coming.

PROF. IGNACIO BARRAQUER (closing): Yo doy las gracias a los distinguidos oftalmologos que me han permitido verificar tan gran numero de operaciones en sus clinicas y a los que han tomado parte en esta discusion, aunque siento no tener tiempo para ser tan extenso como desearia y solo me limitare a esclarecer algunos conceptos sobre mi operacion:

1. No debe llamarse operacion de la catarata por succion toda vez que la ventosa no aspira nada del ojo; unicamente coje el cristalino por su cara anterior.

2. No hay posibilidad de perdida de vitreo si no se practican presiones, aun las mas pequenas, sobre el ojo; toda perdida de vitreo es un defecto de tecnica o de preparacion.

3. No se producen desprendimientos ni de la coroides ni de la retina ya que el vacio vibratorio rompe las fibras de la zonula junto al cristalino, sin que experimenten ninguna traccion en su insercion periferica.

4. Regulando convenientemente la intensidad del vacio, solamente se rompen las capsulas en un minimum despreciable de casos.

5. El Erisifaco es un instrumento delicado y debe cuidarse y probarse cada vez, como hacemos para con el cuchillo de Graeffe.

6. La tecnica de la operacion no es dificil tal como yo la practico; cualquier operador que sepa tallar correctamente un colgajo puede hacerlo, pues considero mas dificil la talla de colgajos correctos que la maniobra de extraccion con el Erisifaco.

7. Es de suma importancia el estudio previo, seleccion y preparacion minuciosa de los pacientes.

Siendo las principales complicaciones en la operacion clasica de la catarata debidas unas a traumatismos del iris y del vitreo por falta de tecnica y otras a la persistencia dentro del ojo de la capsula y restos cristalinianos, es innegable que la extraccion total simple, por un procedimiento que no traumatice ni el iris, ni el vitreo ni ningun otro organo intra-ocular mas que la lesion, ni exija la practica de presiones sobre el ojo, sera el mas aproximado a lo ideal. Con el Erisifaco en nuestra clinica los accidentes operatorios han quedado reducidos a una minima expresion y el tanto por ciento de enfermos con agudeza visual normal ha aumentado considerablemente.

No quiero acabar sin antes invitar a todos mis compañeros a asistir a mis sesiones operatorias en Barcelona.

Apres avoir remercié les ophtalmologues distingués qui m'ont permis d'opérer un si grand nombre de malades dans leurs cliniques, et ceux qui ont pris part a cette discussion, je tacherai de faire remarquer certains points, les plus importants, sur mon opération:

1. On ne doit jamais appeler l'opération "aspiration de la cataracte" toute fois que la ventouse adhère au cristallin mais sans rien aspirer de l'intérieur de l'oeil.

2. La perte de vitré n'est pas possible si on évite la moindre pression.

Chaque fois qu'elle survient c'est à cause d'un défaut de technique ou de préparation du malade.

3. Avec le vide vibratoire qui casse les fibres zonulaires près du cristallin, les tractions de l'Erisiphaque n'arrivent pas à l'insertion ciliaire de la zonule.

4. Pour ne pas casser la capsule il faut régler la quantité de vide selon les conditions physiques de la cataracte à opérer.

5. L'Erisiphaque est un instrument de précision qui doit être soigné minutieusement et essayé chaque fois comme nous faisons avec nos couteaux de Graeffe.

6. La technique de l'opération, telle que je la pratique n'est pas très difficile. L'opérateur qui peut tailler correctement les lambeaux doit l'apprendre facilement; je considère plus difficile la taille du lambeau que la manœuvre d'extraction.

7. J'attache la plus grande importance à l'étude, sélection et préparation des patients.

Les principales complications de l'opération classique reconnaissent deux origines: défauts de technique dans l'acte opératoire et la persistance dans l'oeil de résidus cristalliniens et de la capsule. Certainement l'extraction totale simple, correctement exécutée par un procédé ne traumatisant pas l'iris ni le vitré, serait le plus rapproché de l'idéal.

Dans notre clinique nous avons réduit au minimum les accidents et relevé le pourcentage d'opérés avec acuité visuelle normale depuis que nous pratiquons la Phacoérisis.

J'invite mes confrères américains à assister à mes séances opératoires à Barcelona.

PROF. EMILE GALLEMAERTS (closing): L'opération de Barraquer permettra de réaliser la maximum de résultats. C'est un immense progrès sur tout ce qui a été fait jusqu'ici. Pourra-t-elle être exécutée avec toute la sécurité qu'exige l'opération de la cataracte, l'avenir nous le dira. Nous pourrions au prochain Congrès apporter le résultat des expériences faites dans nos différents pays alors que nous pourrions discuter cette opération au point de vue pratique aussi bien qu'au point de vue théorique.

TROUBLES OCULAIRES CONSECUTIFS À L'OBSÉRVATION DIRECTE DE L'ÉCLIPSE SOLAIRE

DR. DRAGOUTINE KOSTITCH

Belgrade, Jugo-Slavia

HISTORIQUE

Les effets nocifs des rayons directs du soleil sur les yeux sont connus depuis longtemps.

On savait déjà depuis la plus haute antiquité que Denys, tyran de Syracuse, les coupables condamnés à perdre la vue, après leur avoir préalablement enlevé les paupières, les exposait aux rayons directs du soleil jusqu'au moment où leur vision soit complètement abolie. On sait aussi que les Carthaginois employaient le même procédé pour aveugler Attilus Regulus.

Plus tard Galien a observé "des curieux perdre la vue pour avoir examiné avec trop d'attention les taches du soleil."

D'après Beauvais, Galien, Lucretius et Aristote savaient aussi que les éclipses du soleil provoquent les troubles de la vision.

A une période plus rapprochée le célèbre oculiste du XVIII. siècle, signale nettement les effets nocifs du soleil. Après Maître-Jean (1717) Saint-Yves dans son "Nouveau Traité des maladies des yeux" de 1722 dit: "Plusieurs personnes ont perdu leur vue à moitié pour avoir regardé trop longtemps les éclipses du soleil."

A la même époque d'après Boyer, le célèbre naturaliste, Buffon, après avoir regardé longtemps le soleil, fut atteint de mouche volante, et ce trouble de la vue devint insupportable quand ce grand savant examinait les objets très éclairés.

Le cas intéressant de Reid dont l'observation a été reproduite par Mackenzie fut atteint de troubles visuels graves et permanents en 1761 qui sont survenus à la suite de la fixation imprudente du soleil au moment du passage de Venus sur le disque solaire.

Parmi les Auteurs Allemands c'est Plenck en 1783 qui cite aussi quelques cas.

Au XIXème siècle les observations sont plus nombreuses et ont été publiées dans différents ouvrages.

Ces accidents sont cités par Wenzel en 1808 après lui par Demours en 1820 qui a observé "plus de vingt observations d'amauroses in-

complètes survenues peu après l'éclipse du soleil du 7 octobre 1820." C'est à cet auteur qu'appartient le mérite d'avoir trouvé le premier dans ce genre de malade le scotome central, car il cite "mouche fixe par rapport à l'axe optique." Nous avons après les observations citées par Larrey "qu'il relate un cas très grave chez un soldat," par Weller, puis par Sichel (1837) "qui a donné ses soins à cinq personnes qui furent atteintes d'amblyopie de nature torpide chez les unes, irritatifs, chez les autres après avoir trop longtemps fixé le soleil lors de l'éclipse qui a eu lieu en 1886," par Carron de Villard (1836), par Rognetta, par Jaeger (1851) qui a trouvé les troubles oculaires chez quatorze personnes à la suite de l'éclipse solaire, par Van Roosbroeck (1853), par Mackenzie (1857) pour lequel Sulzer affirme que cet auteur a fait le premier la classification de ce genre de troubles oculaires, par Desmarres (1858).

Plus tard ces troubles oculaires sont étudiés dans les mémoires spéciaux en 1879 et 1882 par Dufour, en 1882 par Emmert, Deutschmann, Leber, Haab, l'année suivante par Reich (un cas), Sulzer (4 cas), Swanzy (2 cas).

L'éclipse du soleil du 16 au 17 mars 1887 a donné le mémoire de Haab, Marc Dufour, Sulzer, Emmert et Deutschmann. En 1890 nous avons trois cas de Bock et le cas de Siegfried de Zurich, en 1895 de Duane et Barret et en 1896 de Collins. A cette époque Magavly le premier en 1887 décrit les lésions du fond d'oeil.

A la fin du XIX. siècle nous avons aussi une éclipse du soleil le 28 mai 1900 qui a donné lieu à de nombreuses publications comme celle de Menacho (14 cas), Marquez (2 cas), Aubaret (12), Batten, Siméon Snelle, Lescarret, etc.

Plus tard en 1905 on trouve les observations de Delord et en 1907 celles de Casali (14 observations).

Au XX. siècle à la suite de l'éclipse du 4 avril, 1912, on rencontre les publications suivantes: celles de Blessig (20 cas), Strahoff (112 cas), Jess (33 cas), Katz (6 cas), Hirsch (6 cas), Lasarév (40 cas dont 3 avec rétinite perforante), Bahtin (1 cas), Joffrio (13 cas), Viguodsky (22 cas), Guermann (4 cas), Koubli (8 cas), Tchémolossov (4 cas), Szafinski (11 cas), Cords (32 cas), Hoppe, Uhthoff, Birch-Hirschfeld, etc.

ETIOLOGIE

La fixation plus ou moins prolongée du soleil en general, comme la fixation de l'éclipse de cette grande planete peut provoquer des troubles oculaires.

Pendant l'examen des taches qui apparaissent a la surface du soleil (Galien cité par Demours) et pendant la fixation un peu prolongée du "Passage de Venus devant le disque solaire" (cas de Reid cite par Mackenzie, cas de Swanzy et cas de Duane) peuvent donner les accidents oculaires ce qui confirme nos premiere idées.

Dans les circonstances quand les rayons solaires entrent en abondance dans l'oeil (comme par exemple dans le voyage a travers le champ couvert de neige et le voyage a travers les montagnes glacées) peuvent provoquer ce qu'on appelle "Snow Blindness" remarqué pour la première fois déjà dans la plus haute antiquité par Xenophon cité par Funari. Plus tard les cas avec les accidents pareils sont cités par Maître-Jean et Saint-Yves, Demours, Weller, et Mackenzie.

Reich dans son memoire intéressant de 1880 dans lequel il décrit une véritable épidémie de 73 ouvriers atteints de "Snow Blindness" à la suite du nettoyage de la chaussée transcaucasienne couverte de neige qui passe à travers les plus grandes hauteurs du Caucase. Nous avons encore 15 cas semblables à Samara en Russie et décrits par le Dr. Klykoff en 1904.

A part ces troubles oculaires ci-dessus décrits on trouve souvent des accidents oculaires chez les personnes qui sont obligées de voyager à travers les déserts (Sahara), ou les rayons du soleil sont en abondance.

Avec le développement de l'électricité nous rencontrons les publications dans de différents ouvrages, les lésions rétiniennees survenues à la suite d'un usage du courant électrique.

La retine peut être lésée par les rayons X, substances radio-actives, acétylene, gaz oxyhydrique, les metaux et les verres en fusion, etc.

Malgré qu'on trouve les accident oculaires pendant la fixation à l'oeil nu de l'éclipse solaire, on rencontre aussi ces troubles oculaires chez les personnes qui "En suivant les diverses phases de l'éclipse dans un baquet plein d'eau qui réfléchissait l'image du soleil" (Demours), fixation de l'image du soleil dans un miroir ou dans l'eau (Casali), quand on regarde l'éclipse à travers les doigts (Betten); La fixation de l'éclipse avec les mains en cornet, ou à travers le verre non suffisamment noirci (Villard et Kostitch), à travers le verre irrégulièrement noirci (Demours et Larrey) en fixant à travers le verre coloré par une couleur mal choisie (Berret, James et Snell) à travers une carte à jouer trouée au milieu (Strakhoff), fixation par un télescope imprudemment manié (Lawrance, Regretta, Snell) et enfin à travers un trou pratiqué dans un verre fumé (Strakhoff).

La durée de l'observation de l'éclipse solaire est différente dans différents cas. Dans le cas de Barret la durée de fixation était de 15 minutes, dans celui de Colins de 5 à 10 minutes, dans le cas personnel de 3 à 30 minutes et dans un cas d'un auteur Russe la durée a été de 40 minutes.

Lazareff croit comme la loupe ramasse les rayons du soleil quand on veut à l'aide de celle-ci allumer la cigarette ou un morceau de papier, le cristallin a le même rôle dans le cas de lésion retinienne survenue pendant l'observation à l'oeil nu de l'éclipse solaire par rapport à la macula laquelle doit être brûlée sous l'influence des rayons directs du soleil ramassés par le cristallin et concentrés sur la rétine.

SYMPTOMATOLOGIE

Ordinairement ces "accidents oculaires" qui arrivent comme conséquence de l'examen direct de l'éclipse apparaissent immédiatement après la fixation de ce corps céleste. Mais les victimes de ce phénomène, ordinairement au commencement ne font pas attention à ces "accidents oculaires," en croyant que c'est "la simple prolongation de l'éblouissement habituellement observé quand on regarde trop longtemps ou de trop près un corps incandescent et très lumineux."

Beaucoup de ces malades immédiatement après la fixation de l'éclipse solaire à l'oeil nu se plaignent de voir se promener devant les yeux une tache verte (obs. No. 7) tache sombre comme la poussière (Obs. No. 11) tache jaune (Obs. No. 12) tache jaune verdâtre (Obs. No. 13), une tache qui au début était rouge claire puis violacée et enfin est devenue jaunâtre (Obs. No. 14) et enfin une tache noire (Obs. No. 16). Un malade de Lazareff se plaignait de voir un "cheval vert."

Il y a certains malades qui ne voyaient pas les mots entiers pendant la lecture, d'autres perdaient certaines lettres et en cherchant à les voir pendant la lecture ils, arrivaient finalement mais perdaient les lettres voisines.

D'autres malades se plaignaient que certaines lettres leur paraissaient être couvertes par une espèce de tache d'encre.

Une de nos malades se plaignait qu'elle voyait son mari mais n'apercevait pas "le milieu de la tête."

Un autre de nos malades (Obs. No. 4 or 13) qui depuis l'éclipse d'avril de cette année se plaint que la vision de son oeil gauche est troublée, mais il accuse d'avoir pendant la lecture un point noir, qui

l'empêchait de voir deux ou trois lettres de chaque mot qu'il lisait et au lieu de ces lettres il voyait une tache bleu verdâtre dont le vert était très foncé qui ressemblait à une tache d'encre.

L'Etat du Fond D'Oeil.—A l'ophtalmoscope on décele des lésions du fond d'oeil provoquées par la fixation à l'oeil nu plus ou moins longtemps de l'éclipse solaire. Ces lésions pourtant, surtout immédiatement après la fixation sont souvent malgré les troubles de la vision introuvables par l'ophtalmoscope et le fond d'oeil paraît tout-à-fait normal comme dans les cas de Emmert, Swanzy, Bocca, Duane, Snell, Lawward, Aubaret, Menacho et observations personnelles.

Comme nous venons de le dire plus haut quand la lésion du fond de l'oeil existe, on voit à l'ophtalmoscope quelque fois la coloration plus intense de la tache jaune (Reich, Schirmer) d'autres fois on remarque une pigmentation plus intense de cette région (Sulzer et Dufour) ou l'oedème de macula. Mais de tous ces changements du fond d'oeil on voit le plus souvent au niveau de la macula des taches rouges noires ou de la couleur des framboises, ce qui prouve qu'il y a une congestion plus ou moins intense dans cette région (Swanzy, Bocca, Aubaret, Menacho, Delord, Vigodsky, Joffrio, Bourzeff).

Les lésions vues à l'ophtalmoscope peuvent être quelquefois plus intenses que celles que nous venons de citer plus haut. Lazareff a trouvé chez trois malades après une fixation longue a 1 (oeil nu de l'éclipse solaire, la perforation de la retine au niveau de la macula.

PATHOGENIE

Nombreux sont les auteurs qui ont fait différentes expériences par les rayons solaires sur les yeux de différents animaux pour expliquer les lésions de l'oeil, que l'on trouve chez l'homme que dans les troubles provoqués par l'éclipse solaire.

Parmi ces auteurs c'est Czerny (1867) a le premier commencé par les expériences pareilles sur les animaux, après lui c'est Deutschmann (1882), Widmark (1890), Aubaret et Lescaret (1907) et Birch Hirschfeldt (1912).

Czerny et Deutschmann après avoir préalablement dilaté la pupille d'une grenouille et d'un lapin concentraient à l'aide d'un miroir concave ou d'une lentille convexe des rayons du soleil sur la rétine des animaux en expérience.

Après l'effet de dix à quinze minutes de ces rayons ils observaient sur la retine à l'aide de l'ophtalmoscope une tache claire entourée par une

auréole jaunâtre ou grisâtre et un peu plus tard la couleur d'auréole devenait d'abord rougeâtre puis brune et enfin presque noire.

Pendant trois semaines le processus de cette lésion évoluait, les couleurs changeaient et finalement il se formait au centre du foyer rétinien une tache durable.

Au bout d'un certain temps on énucléait les yeux en expérience et après un examen histologique on constatait que ce sont les couches des fibres nerveuses, des cônes et bâtonnets, et la couche des granulations internes qui subissent le plus de dégâts.

Dans ces couches ci-dessus citées, il se forme une dégénérescence granulaire avec un développement hypertrophique des cellules fibrineuses.

Il est difficile d'adapter l'œil expérimenté pour que les rayons solaires tombent juste sur les fovea, sur la macula. Puisque la macula est composée surtout de cônes et bâtonnets et couches granuleuses externes, il est compréhensible que les lésions sur ce lieu soient d'un caractère sévère (Lazareff).

Strahoff, croit que les cônes et bâtonnets paraissent être plus lésés à cause d'une couche pigmentaire qui se trouve derrière et qui attire beaucoup les rayons lumineux.

Birch-Hirschfeld a exposé les yeux de lapins aux rayons solaires dans les conditions qui se rapprochent le plus aux conditions de l'éclipse solaire.

Par les recherches microscopiques déjà expérimentées il a trouvé une hyperémie choroidale, l'œdème de la couche pigmentaire et la dégénérescence de la partie externe de la rétine.

Dans ces expériences de l'exposition des yeux de lapins aux rayons ultra-violets Birch-Hirschfeld est arrivé aux résultats contraires de ses premières expériences, il a trouvé que la partie externe de la rétine était intacte mais la couche des cellules ganglionnaires et la couche granuleuse interne étaient lésées. Les rayons ultra-violets sont absorbés par la couche interne de la rétine, mais les rayons lumineux traversent cette couche et ne sont absorbés que par la couche pigmentaire, par conséquent les dégâts de la rétine qui dépendent de l'influence des rayons solaires pendant l'éclipse se résument par une nécrose coagulente provenant de l'épithélium de la vision et par exudation de la choroïde.

De ces expériences on voit que les rayons calorifiques ne sont pas nuisibles comme les rayons lumineux.

Aubaret et Lescaret ont fait quatre expériences sur des lapins, en

exposant durant 20 à 40 minutes les yeux de ces animaux aux rayons calorifiques, l'autre aux rayons chimiques et les yeux des deux derniers aux rayons solaires.

Ces auteurs sont loin des résultats de leurs devanciers. Après l'examen ophtalmoscopique immédiat et les jours suivants ils n'arrivent pas à déceler les lésions apparentes. A l'examen microscopique après énucléation de l'oeil expérimenté ils n'ont pas pu trouver la partie éblouie de la rétine. Aubaret dans son travail publié dans les archives d'ophtalmologie du février 1907 se borne à critiquer le procédé de l'expérimentation de ses devanciers en abolissant leurs théories que la radiation calorifique et chimique peuvent produire photo-trauma. Pour lui la radiation lumineuse seule produit le photo-trauma.

SCOTOME CENTRAL

C'est Emmert qui a donné le nom de scotome héliéclipticum et le nom de scotome hélioplégicum est donné par Szafinsky.

Dans les incidents oculaires provoqués par l'éclipse on trouve presque toujours un scotome qu'on nomme ordinairement scotome héliéclipticum ou scotome hélioplégicum. Quelquefois on ne trouve pas de scotome ou même s'ils existent ils sont si petits qu'on ne peut pas les définir par le périmètre ordinaire et ces scotomes peuvent être psychiquement annulés par les malades.

Le scotome est le plus souvent central et les cas de scotome para central sont plus rares.

Dans certaines observations publiées on trouve les scotomes "positifs" comme une tache éblouissante et dans l'autre le scotome "négatif" c'est à dire le scotome qui se manifeste par un petit déficit dans le champ visuel. Quand à la forme de ce scotome c'est la forme circulaire "Ring scotome" qui est la plus habituelle. Par les examens scotométriques (Jess) on est arrivé à conclure que la forme du scotome correspond au optogramme du soleil est rare (Birch-Hirschfeld).

La forme de ce scotome central est quelquefois ovale.

Dans un cas curieux de Duane relatif à l'observation du passage de Vénus devant le soleil, ce scotome avait une forme circulaire avec un échancrure à la partie supérieure et reproduisait exactement la forme du disque solaire au moment de l'observation.

Le scotome d'après Aubaret consiste dans un edème locale de la rétine et d'après Villard correspond à une hémorragie locale de la rétine.

L'influence de l'éclipse solaire sur l'acuité visuelle.—L'acuité visuelle

chez les malades qui ont des accidents oculaires survenue à la suite de fixation à l'oeil nu de l'éclipse solaire, peut être de 0.8–0.9, ou l'acuité peut être normale. Mais il y a des cas dans lesquels l'acuité visuelle est de 0; 1 à 0.2.

Les cas où la vision est presque normale s'expliquent par le fait que la lésion de la rétine n'occupe pas toute la fovea centrale de la macula.

Dans certains cas le malade peut améliorer sa vision par une fixation excentrique c'est à dire par une déviation de l'oeil pendant le regard, et par ce fait l'objet n'est pas fixé par le centre de macula, mais il est fixé par les autres parties de macula qui ne sont pas si lésées que la fovea centrale. Par cela on peut conclure en dehors de centre de la macula que les autres parties de celle-ci restent intactes (Strahoff).

LE RAPPORT ENTRE LES TROUBLES ET LA REFRACTION

Théoriquement les yeux des émmétropes sont plus exposés aux lésions pendant la fixation que ceux des hypermétropes et myopes.

Dr. Tchémolossoff de Pétrograd a publié un cas qui correspond à l'hypothèse ci-dessus.

Une jeune femme bien portante a fixé avec les deux yeux nus l'éclipse solaire; elles n'a eu les troubles oculaires qu'à un seul oeil qui était émmétrope. L'autre oeil dont la réfraction était myopique est resté sans aucun accident.

Nous avons aussi comme chez notre malade obs. No. 18 l'oeil émmétrope était lésé et l'autre oeil hypermétrope resta intact.

COMPLICATION

Marquez a publié un cas dans lequel aussitôt après la fixation à l'oeil nu de l'éclipse solaire, il a trouvé ophtalmoscopiquement une grande congestion des papilles et neurorétinite dans les deux yeux. Les atrophies des nerfs optiques arrivent ordinairement comme complication tardive.

Comme nous l'avons déjà remarqué Lazareff a trouvé trois cas dans lesquels il a constaté, bientôt après la fixation, la perforation de la rétine au niveau de la région maculaire.

Batten a trouvé un cas de thrombose d'une artère rétinienne et un autre cas d'hémorragie de la rétine.

Menacho a eu l'occasion de voir 14 malades qui ont présenté différentes complications de l'oeil survenues à la suite de l'éclipse solaire.

De ces 14 observations un malade présentait une papillite centrale, 2 malades présentaient de l'hyalite, 1 cas d'apoplexie maculaire, un autre

une nevrite rétro-bulbaire; une fois il a trouvé chez un malade de la lymphangite antérieure avec glaucome et enfin deux fois des nevrites optiques.

Sulzer a trouvé aussi un cas de neuro-rétinite.

Stéphenson a constaté un cas de nevrite légère.

Katz de ces 6 observations a trouvé un cas de conjonctivite avec kératite du limbe. (2^e) cataracte polaire postérieure; (3^e) paralysie du sphincter pupillaire.

Marenholtz a trouvé un cas de nevrite optique.

Hirsch de 6 cas il a remarqué chez trois malades de conjonctivite.

Jssakowitz a observé un cas de l'éblouissement du cristallin. Nous avons trouvé aussi un cas de conjonctivite (obs. No. 19).

Aubaret et Villard pensent que les complications graves survenues à la suite de la fixation de l'éclipse sont rares et arrivent à l'hypothèse, à part des rayons solaires qu'il y a d'autres causes qui provoquent ces troubles oculaires.

Ainsi Aubaret pense que la complication d'hyalite chez des malades citée par Menacho n'est pas survenue à la suite de fixation de l'éclipse, mais qu'elle est survenue à cause de la myopie forte, qui existait chez ces malades déjà avant l'éclipse.

Villard a publié une observation d'un malade fortement myope chez lequel il a trouvé un décollement de la rétine à la suite de fixation de l'éclipse. Cet auteur croit que cette complication n'est pas arrivée comme conséquence de l'action des rayons directs du soleil.

Fromaget a constaté chez un malade à la suite de l'éclipse une choroidite séreuse, mais ce malade souffrait pendant 6 ans avant l'éclipse de sinusite maxillaire du même côté.

PRONOSTIC

Dans cette maladie le pronostic doit être toujours réservé. Il y a des cas qui guérissent assez rapidement, dans lesquels l'acuité visuelle devient presque normale ou tout à fait normale. Le petit scotome qu'on rencontre chez ces malades disparaît aussi assez rapidement. Dans d'autres cas l'acuité visuelle s'améliore seulement au bout de 4 à 6 mois et elle n'aboutit que jusqu'à 0.5 à 0.6. Le scotome ne disparaît pas chez ces malades, à cause de la disparition des cônes et des bâtonnets au niveau du centre de la macula qui sont "brulés" par les rayons directs du soleil. Malheureusement comme ces éléments ne se régénèrent pas, ces malades sont condamnés à supporter ce scotome éternellement.

Dans certains cas le pronostic est encore plus grave. "Maitre Jean fait allusion: toutes ces choses disparaissent quand les fibres de la rétine se remettent dans leur état normal; mais quand le vice que ces fibres ont contractée est considérable il continue quelquefois à paraître tout le reste de la vie."

Saint-Yves signale: "Des personnes qui ont perdu la vue à moitié pour avoir regardé trop longtemps des éclipses du soleil."

Rognetta cite un cas qui resta complètement aveugle.

De Wecker croit que l'abaissement de l'acuité visuelle et le scotome peuvent rester éternellement.

Reich a constaté un cas où l'acuité visuelle est resté à 0.3 pour toujours avec scotome central persistant pour les couleurs.

Dans le cas de Winsellmann l'acuité visuelle a été presque nulle pendant 3 ans à partir du commencement des accidents oculaires.

Dans l'observation de Collins: "Les troubles de l'oeil le plus atteint n'avaient pas sensiblement diminués 9 ans plus tard."

Duane cite un cas dans lequel le scotome existait pendant 12 ans après le début des accidents, avec la complication d'attaque d'hémi-anopsie.

Aubaret a constaté chez un malade après 45 ans de l'éclipse solaire un scotome héliéclipticum, avec une diminution de l'acuité visuelle de $\frac{2}{3}$ et dans un autre cas il trouva un scotome après 28 ans de l'éclipse solaire.

TRAITEMENT

La plupart des auteurs conseillent au point de vue prophylactique de ne regarder le soleil qu'à travers des verres fortement teintés, qui atténuent beaucoup l'intensité des rayons qu'il émet. Les verres employés doivent être suffisamment teintés en noir. Autrement, comme on l'a déjà constaté, si l'on fixe l'éclipse solaire à travers le verre insuffisamment teinté; dans ce cas les troubles oculaires peuvent se produire, aussi bien dans le cas de fixation à l'oeil nu.

Certains auteurs comme Holden ont conseillé de superposer trois verres, un rouge, un vert et un bleu. Il paraît que ces verres colorés bien choisis "ne changeraient en rien la couleur du soleil, tout en affaiblissant suffisamment son intensité lumineuse pour en rendre l'observation directe inoffensive pour l'oeil."

Quand le malade s'adresse au médecin immédiatement après les accidents, il faut mettre les yeux du malade au repos complet. Le malade ne doit pas quitter la chambre; laquelle doit être peu éclairé; on ordonnera un purgatif que l'on renouvellera plusieurs fois; on

prescrira des saignées locales (vantouses scarifiées de Heurteloup), on peut conseiller aussi l'injection de strychnine; enfin plus tard on donnera de l'iodure de potassium.

A part ce traitement nous avons conseillé à nos malades qui étaient presque tous des élèves du Lycée, de quitter l'école provisoirement et de passer leurs vacances à la campagne dans la verdure.

Le plus part de nos malades ont acceptés notre conseil, d'autant plus que l'éclipse solaire du 9 avril est tombé juste avant la fin de l'année scolaire.

LES OBSERVATIONS

Obs. No. 1.—Zagarka Popovitch, âgé de 23 ans. Bonne.

Le 9 avril elle a fixé avec les deux yeux l'éclipse solaire à 10 heures du matin. Après une courte fixation des deux yeux elle a continué à fixer pendant une demi-heure avec l'oeil droit seulement. Tout de suite après la fixation elle a perdu la vision momentanément, mais aussitôt la vision est revenue surtout à l'oeil gauche, et la vision s'est améliorée difficilement, mais comme nous verrons plus tard l'acuité visuelle surtout de l'oeil droit est resté en défaut.

13 avril 10 D. Devant cet oeil elle a une tache qui lui a troublé cet oeil. La couleur de cette tache au début était jaune puis verte et enfin est devenue rouge. La malade a déclaré qu'avec l'oeil droit elle voyait son mari mais elle ne lui voyait pas le milieu de la figure. Son acuité visuelle a été le 13 avril de 0.7. Donc chez cet oeil existe le scotome central.

A l'examen ophtalmoscopique on voyait que l'auréole de la macula était ébloui, et dans le centre de la macula on voyait un petit cercle de couleur foncée et vers la périphérie de ce centre il existait une petite tache jaune.

O. G. Pas de scotome. F. O. normale V. = 0.9.

A la sciascopie les deux yeux étaient émétrôpes.

19 avril O. D. Sans changement. O. G. V. = 1.

Obs. No. 2.—A. D. élève du Lycée. Il a fixé l'éclipse du 9 avril 2 fois avec l'oeil droit nu en fermant l'oeil gauche. Le lendemain il a remarqué que sa vision n'était pas si bonne qu'avant, et surtout une tache grisâtre persistante devant cet oeil le gênait.

Le 12 avril. O. D. V.: 0.5. En lisant il ne voit que chaque troisième lettre du mot F. O. macula avec un auréole clair, dans le centre de la macula une petite tache rouge foncée. Petit scotome central. O. G. normal. A la Sciascopie les deux yeux sont émétrôpes.

4 octobre, même année. O. D. V.: 1. Disparition de la tache. F. O. La petite tache de la macula est diminuée. Pas de scotome.

Obs. No. 3.—F. F. Agé de 14 ans, élève du Lycée a fixé le 9 avril, 1921, avec l'oeil gauche nu l'éclipse solaire pendant 4 à 5 minutes. Aussitôt il a remar-

qué que la vision de cet oeil était troublée et en même temps une petite tache persistante se promenait devant cet oeil, pendant la lecture il ne voyait pas certaines lettres du même mot.

14 avril. O. D. normal. O. G. F. O. Toute la région de la macula était couverte on dirait par une sérosité qui empêchait de voir les détails de cette région. Scotome central très grand. V.: 0.3.

20 septembre. O. G. F. O. Au niveau du centre de la macula une tache gris foncé. Scotome existe encore. V.: 0.6. O. D. normal.

Obs. No. 4.—M. V. élève du Lycée, âgé de 17 ans, a fixé le 9 avril au début avec les deux yeux à travers le verre fumé l'éclipse solaire. Au bout d'un certain temps il a continué à fixer avec l'oeil gauche nu à peu près pendant 20 minutes. Son oeil droit pendant la fixation était fermé. Aussitôt il a remarqué un éblouissement et la fatigue des deux yeux. Cet éblouissement n'est pas été de longue durée, mais devant son oeil gauche, persistait une tache l'empêchant de voir deux ou trois lettres de chaque mots. A la place de ces lettres il voyait une tache de la couleur bleu verdâtre-foncé.

11 avril. O. D. normal. O. G. V.: 0.4. F. O. Dans le centre de macula on trouve une tache en forme de demi cercle de couleur rouge foncé. Scotome central positif.

20 avril. O. D. normal. O. G. V.: 0.7. Le reste sans changement.

1 octobre. O. D. normal. O. G. Scotome central positif V.: 0.7. F. O. Petite tache blanche dans la région maculaire.

Obs. No. 5.—Le nommé M. V. élève du Lycée âgé de 16 ans a fixé avec l'oeil gauche nu l'éclipse solaire du 9 avril 1921. Aussitôt il a remarqué une tache devant cet oeil qui lui cachait une partie de petits objets. Cette tache a duré pendant 2 jours. Nous ne l'avons vu que 7 jours après l'accident. Il n'avait pas de scotome. O. D. normal. O. G. le contour du macula n'était pas très net. L'acuité était normale aux deux yeux.

2 septembre. Les deux yeux étaient normaux.

Obs. No. 6.—Le nommé M. Man, âgé de 11 ans élève du Réal gymnase. Ce malade a fixé l'éclipse solaire du 9 avril deux fois avec les deux yeux nus. Chaque fois la durée de fixation était de 10 à 15 minutes. Après la fixation il est rentré en classe pour travailler, mais quand il a commencé à lire toutes les lettres lui semblait être couvertes par des points noirs ou plutôt comme il disait couvertes de "taches d'encre." En même temps les lignes imprimées lui semblaient être interrompues par places. Nous l'avons vu le lendemain de la fixation c'est-à-dire le 10 avril, 1921.

O. G. F. O. Tout le centre de macula était couvert par une tache rouge fortement foncée. A la partie inférieure de cette tache on voyait un petit point jaune comme une tête d'épingle. L'auréole de la macula était fortement hyperémie. V.: 0.2.

O. D. F. O. Tout l'auréole de la macula était oedemaciée et la partie in-

férieure était presque bleuâtre. Au centre de macula existait une tache fortement rouge. V.: 0.3. Le soleil le gêne beaucoup. Le scotome central des deux yeux était positif.

10 octobre, même année. Le scotome positif des deux yeux O. G. V.: 0.2. F. O. auréole maculaire pas nette. Au centre de la macula on voit un petit cercle rouge avec un point blanc au centre. O. D. V.: 0.3. F. O. auréole maculaire pas nette.

Le malade lit un peu plus facilement mais encore assez difficilement de telle sorte qu'il était obligé de repasser ses examens à la session de novembre.

Obs. No. 7.—R. B. élève du Lycée âgé de 10 ans. Il a fixé pendant 2-3 minutes avec les deux yeux l'éclipse solaire. Le même jour il avait devant les deux yeux une tache verdâtre. Au bout de quelques jours cette tache avait disparue. Nous l'avons vu le 12 avril. Pas de scotome. Les taches persistent. F. O. Aux deux yeux normaux ainsi que l'acuité visuelle.

Obs. No. 8.—M. S. élève du Lycée, âgé de 13 ans. Le malade a fixé avec l'oeil droit pendant 3-4 minutes, l'éclipse solaire du 9 avril, 1921. Il y a eu une tache devant cet oeil aussitôt mais elle a disparue le lendemain. Les lignes et les lettres lui semblaient être courbés. Nous l'avons vu le 11 avril, 1921. O. D. V.: 1 F. O. normal.

Obs. No. 9.—B. M. élève du Lycée, âgé de 12 ans. A fixé plusieurs fois dans la matinée du 9 avril l'éclipse solaire avec l'oeil droit tantôt nu, tantôt à travers le verre fumé. Le même jour il avait une tache bleu-verdâtre devant cet oeil. En lisant il ne voyait pas certaines lettres de chaque mot. Nous l'avons vu le 12 avril. Ce malade présentait à l'oeil gauche un petit scotome. V.: 0.9. F. O. hyperémie légère de la macula.

10 août. O. D. V.: 1. Pas de scotome. F. O. normal.

Obs. No. 10.—J. C. Elève du Lycée, âgé de 13 ans, a fixé avec les deux yeux l'éclipse solaire du 9 avril, plusieurs fois pendant 2-3 minutes. Dans la même journée il a remarqué devant son oeil gauche une tache; pourtant devant l'oeil droit qui est strabique n'a rien remarqué. Il est venu chez nous le 10 avril 1921.

O. D. V.: 0.1 Pas de scotome. Strabisme convergent. F. O. normal. Pas de tache subjective. O. G. V.: entre 0.9 et 1. Pendant la lecture les lignes sont troubles. La tache persiste. Petit scotome central.

Obs. No. 11.—A. A. âgé de 14 ans, a fixé le 9 avril avec l'oeil droit 3 fois l'éclipse solaire. Le même jour il a remarqué une tache devant cet oeil qui l'empêche de lire. Cette tache lui semble comme une petite poussière. Le 11 avril il est venu chez nous. O. D. V.: 0.6. F. O. A l'ophtalmoscopique on ne voyait pas bien l'auréole. Au centre de macula il existait un petit point rouge sous la forme d'un petit cône découpé. Le scotome central existe. O. G. normal.

Obs. No. 12.—S. R. A fixé avec l'oeil droit et il a fermé son oeil droit strabique. Il a fixé en deux fois pendant 4-5 minutes. Après la seconde fixation il a remarqué une tache jaune devant son oeil droit. Le 15 avril à la date où nous l'avons vu pour la première fois son oeil droit présentait l'acuité visuelle de 0.7. La tache jaune persiste. F. O. normal. Petit scotome. O. S. Strabisme convergent. V.: 0.5. Pas de tache.

20 août, 1921. O. D. Pas de scotome. Pas de tache. V.: 1 F. O. normal.

Obs. No. 13.—S. Y. âgé de 12 ans. A fixé le 9 avril l'éclipse solaire pendant 15 minutes avec l'oeil gauche tantôt nu tantôt à travers le verre fumé. Le lendemain après la fixation il a remarqué devant son oeil gauche une tache jaune-verdâtre. Pendant la lecture il ne voyait pas certaines lettres et surtout le soir quand il lisait à l'éclairage électrique.

13 avril. Il est venu pour la première fois à la consultation chez nous. O. G. V.: 0.4. Scotome existe. La tache persiste. F. O. Au centre de macula une tache presque noire, avec deux petits points jaunâtres au centre. O. D. normal.

Obs. No. 14.—T. P. âgé de 20 ans. A fixé le 9 avril avec O. D. pendant une heure avec interruption tantôt à l'oeil nu tantôt à travers le verre fumé. Aussitôt il a remarqué devant son oeil droit une tache rouge, le lendemain cette tache est devenue violacée et le 13 avril le jour où il est venu chez nous pour la première fois cette tache était de couleur jaune. O. D. V.: 0.7-0.8. F. O. auréole de la macula très pâle. Au centre de la macula une tache en forme de bague avec un petit point jaune au centre. Scotome existait. Pendant la lecture le malade ne voyait pas les mots entiers.

1 Sept. Sans changement.

Obs. No. 15.—B. M. élève au Lycée, âgé de 18 ans, a fixé le 9 avril à travers le verre fumé, puis avec l'oeil droit nu pendant 10 minutes. Le même jour il a remarqué devant son oeil droit une tache jaune pâle. En lisant il ne voyait pas certaines lettres. Le 19 avril nous l'avons vu. O. D. F. O. Au centre de la macula une petite tache rouge sous le point jaune au centre. Scotome paracentral. La tache qui a persisté devant cet oeil est moins forte. V.: 0.8. O. G. normal.

Obs. No. 16.—M. S. élève du Lycée, âgé de 13 ans, a fixé le 9 avril 1921 l'éclipse solaire pendant 15 minutes avec l'oeil droit nu. Le même jour il a remarqué une tache noire devant cet oeil; et pendant la lecture il ne voyait pas certaines lettres du mot.

19 avril. Le jour où nous l'avons vu il présentait les symptômes suivants: O. D. Scotome existe. F. O. Au centre de la macula on voyait un petit point rouge. Au niveau de la partie supérieure de l'auréole existait un point rouge foncé et entre ce point et celui du centre il y avait deux points blancs. V.: 0.5. O. G. normal.

4 Octobre. O. D. Scotome persiste. V.: 0.6. F. O. Au centre de la macula un point blanc au dessous duquel on voyait une tache rouge en arc. La lecture est possible. O. G. normal.

Obs. No. 17.—S. V. élève du Lycée, âgé de 13 ans. A fixé le 9 avril avec les deux yeux nus pendant 3 minutes. Le lendemain il a remarqué une tache noire devant chaque oeil ce qui l'a fait venir le 11 avril à la consultation chez nous. O. D. Pas de scotome. V.: 1 F. O. On n'a remarqué rien d'anormal à l'ophtalmoscopie. O. G. La même chose que l'autre.

25 avril. Les scotomes subjectifs ont disparus.

Obs. No. 18.—B. K. élève du Lycée âge de 12 ans a fixé le 9 avril avec les deux yeux nus pendant une demi heure. Le même jour il a remarqué devant l'oeil gauche une tache verdâtre. Le 14 avril jour où nous l'avons vu pour la première fois. O. G. A la sciascopie -2 D. Scotome central existait. V.: 0.1. Avec la correction de $+2$ D. l'acuité était de 0.4. La tache était si grande qu'elle l'empêchait de voir plusieurs lettres du mot. F. O. Dans un petite veine qui passe en dessus de la macula à l'image renversé on remarquait un petit noeud. Toute la région maculaire oedémacié. A la partie supérieure du centre de la macula il existait une petite tache de la couleur framboise.

O. D. émmétrope. V.: 1. F. O. normal. Il n'est plus revenu a la consultation.

Obs. No. 19.—P. K. élève du Lycée âgé de 14 ans a fixé avec l'oeil droit pendant plus d'une demi heure. Nous l'avons vu le lendemain de l'éclipse. Il se plaignait que cet oeil avait des larmoiements et en même temps d'avoir du sable sous les paupières et en particulier sous les paupières supérieures. O. D. Conjonctivite sub aigue. Cette conjonctivite l'empêchait de remarquer la tache noire habituelle chez ce genre de malades, mais après la guérison de la conjonctivite, la tache noire est apparue. V.: 0.9. F. O. la région maculaire un peu rougeatre. Pas de scotome. O. G. normal. Il n'est plus revenu à la consultation.

Obs. No. 20.—J. K. élève du Lycée, âgé de 11 ans, a fixé avec l'oeil droit nu pendant 5 à 6 minutes. Une heure et demi après la fixation il a remarqué une espèce de brouillard jaunâtre. Ce brouillard comme une toile d'araignée l'empêchait de lire. Le lendemain de la consultation il est revenu de nouveau chez nous. Nous l'avons constaté à la sciascopie une myopie de 5.0 aux deux yeux O. D. V.: après la correction 0.6. F. O. Dans la région maculaire oedemacié. Scotome central. Le brouillard persiste. O. G. myopie de 5.0. V.: après la correction: 1. F. O. normal.

25 avril. Aucun changement.

CONCLUSIONS

1. L'observation de l'éclipse solaire du 9 avril 1921 à Belgrade a provoqué chez les curieux qui ont fixé l'éclipse avec les yeux nus des troubles oculaires, forts chez les uns, plus faibles chez les autres.

2. Il est caractéristique que, de nos 20 malades, 19 sont des élèves des Lycées de Belgrade.

Avant l'éclipse, les lycéens de Belgrade étaient prévenus du danger que courent leurs yeux s'ils ne prenaient pas les précautions nécessaires, qui ont été expliquées par nous et par leurs professeurs.

3. Nous croyons que les élèves, empêchés de lire par des éblouissements provoqués par scotome, sont venus immédiatement consulter le médecin, tandis que les autres malades qui, surtout chez les paysans, croyaient que ce n'étaient que des troubles passagers, ne sont pas venu consulter le médecin.

4. Les lésions oculaires provoquées par la fixation de l'éclipse solaire peuvent être faibles ou très fortes.

5. Le symptôme le plus habituel chez ces malades est le scotome hélioplégicum ou héliéclipticum.

6. Il semble que les rayons lumineux directs du soleil sont les plus nuisibles.

7. Les yeux émmétropes sont plus exposés aux accidents pendant la fixation que les yeux myopes ou hypermétropes.

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DISCUSSION

COL. R. H. ELLIOT (London, England): I saw a good deal of eclipse blindness in India, and would like to mention some additional causes which are not mentioned in the paper. One is the reflection of light from water when a boat is being steered in the sun's eye on a tropical sea or river; another is taking observations by the sextant at sea; a third is a religious rite of the Brahmans: Every orthodox Brahmin has to cross his hands and look through them at the sun at certain times of day. I understand a number of them do not trouble to look at the sun, but look through a wall on which the sun shines; but a number do really look at the sun, and Col. Kirkpatrick has described eclipse blindness as a result of this rite. The practice of observing an eclipse through a pail of water is common in the Tropics, and is extremely dangerous. It may lead to very definite eclipse blindness.

Some little time ago I saw an officer of the Indian Government who had ridden a long distance besides rice fields in the early morning. The sun came obliquely through the rice fields to his eyes, and when he came to me afterward he had a well-marked eclipse scotoma.

An interesting point in Dr. Kostitch's paper is the youth of the subjects, because the opinion hitherto held by ophthalmologists is that it is rare to find eclipse scotoma in young people.

A point I do not find referred to is the association of eclipse scotoma with a ring scotoma lying between 35 degrees to 50 degrees in the field. This has been noticed both in aviators and in those who have had to do with anti-aircraft guns.

Another extraordinary thing in this paper is the number of people in whom both eyes are affected, because past experience would seem to indicate that most of the patients had involvement of only one eye.

There is a practical point I would like to call attention to in the differential diagnosis from hysteria. Scotoma due to sun-blindness is small; at twenty feet away it just covers the face. I recently saw a girl who had looked at the sun, and believed she had central scotoma, but her scotoma covered the whole subject standing a few feet away. That was obviously not a central scotoma, as the subsequent history proved. Another point in hysterical cases is that the scotoma does not necessarily appear immediately after the exposure to the sun, whereas in the real cases the patients give a history of a scotoma coming on at once.

A point about prophylaxis. The best way to look at a sun eclipse is first to use a negative which has been suitably exposed to light and fully developed so as to get a uniform dark plate; and second, to avoid looking at the sun for any prolonged period. It is the prolonged looking at the sun that does the mischief. Another point is to do what certain countries—Sweden, for instance—have done, and to start a press campaign before each eclipse warning people of the dangers involved in looking at the sun, telling them how they may safely and comfortably view the eclipse.

DR. GEORGE MACKAY (Edinburgh, Scotland): The subject under discussion attracted my attention many years ago. The writer of today's paper has limited his survey of the literature to the period of 1900 to 1912, which makes me feel somewhat antediluvian, because my paper was written in 1894, and has not received Dr. Kostitch's attention. However, I am somewhat to blame because the title of my paper did not indicate that it had reference to eclipse blindness, it being entitled "Blinding of the Retina by Direct Sunlight." It so happens that a majority of cases of retinal damage from solar rays do arise after gazing at an eclipse, but I venture to think that my title is more correct, since this damage may happen at any time if an observer directs his gaze to the sun's face. My paper contained a complete survey of the literature so far as I knew up to 1894. I was about to make a fresh survey of the whole literature of the subject about 1912, as I thought a sufficient interval had elapsed, but just then there appeared a paper written by a German, a monograph presented I think as a thesis at one of the German universities, and it so completely covered the ground that I set to work to translate that rather than prepare a new paper.

I have already pointed one moral, namely, that if you want to attract the public eye you must be careful what title you employ, for my paper has been frequently overlooked.

Another point I would like to make is this: I would most respectfully suggest to the organizers of meetings such as this that before they put out a pre-session volume they should endeavor to anticipate that by a still earlier pre-session list of the titles of the papers which will be discussed. That could be sent out before the full contents of the papers are set up in print. Those who have come from a distance are at a great disadvantage in being uninformed before leaving home as to the subjects to be discussed. I must make apology, therefore, which is not altogether my own fault, that I cannot remember the name of the writer of that important paper, but it will undoubtedly be found in the *Index Medicus* about the years 1912 or 1913. It is a most admirable monograph, bringing the literature up to that date.

There are one or two points to which reference has not been made by Dr. Kostitch, and on which I should like to have had his opinion had he been here. One symptom to which he does not refer, but which many have observed, is a quivering movement complained of by the patient. What is that due to? It passes away in many cases, but it is often persistent. In fact, in one case, that of a medical colleague, it is the only thing he still feels, although the accident happened a quarter of a century ago. I suggest that whereas the normal stimulus of moderate light only excites molecular or atomic vibrations in retinal cones and rods, the more violent assault of focussed sunlight loosens the attachment of some of the cells to one another so that a whole cell vibrates separately or in groups.

Another question is as to whether the injury is produced by the concentration of the light rays or from the heat rays. One is inclined to think that it is more probably the light rays than the heat rays. That is a point upon which I should be glad to have had some information.

Another point upon which I especially endeavored to start inquiry in my communication of 1894 has reference to the prognosis. Recently a book was sent me for review, "Medical Ophthalmology," by Dr. Foster Moore, and I am gratified that he has reproduced the tentative conclusions which I put forward in 1894. It appeared to me that here we had an illustration of the most exact injury to the human tissues which it was possible to produce by purely physical methods; an injury to a part which is beyond external contamination by septic influence. Moreover, the injured part can give expression to the extent and intensity of the damage received, and continued observations afford a very exact method of indicating the prognosis, dependent upon the nature and extent of the primary injury and the time elapsing since it was received. The basis for deduction is broadened if there is any record of the vision prior to the sun blinding. Therefore what I endeavored to lay down was this, based upon the results up to 1894. I defined the term "practical recovery" as meaning the "cessation of obtrusive defect" which leaves the patient unconscious of having received damage. I was able to classify the cases into four groups. Taking direct acuteness of one-third of Snellen's type as the starting point, the patient with one-third vision in the first week has a good chance for practical recovery in one month; the patient with one-third vision in the second week has a good chance for practical recovery in three or four months; the patient with one-third vision in the third week has good chance for practical recovery in five or six months, but

if the vision at any time is less than one-third of the normal there is a very poor chance of ever recovering six-sixths. I venture to hope that these suggestions may be pursued and confirmed or corrected.

DR. F. M. FERNANDEZ (Havana, Cuba): With each new eclipse the number of cases of retinal lesions becomes less and less. This is due in Cuba to the extensive warnings that are published in the daily papers.

Some few cases of solar retinitis have been observed in Cuba in the past fourteen years. Symptoms have been moderate in the majority of the cases—central and para-central scotoma, diminution of visual acuity, and so forth. There have been observed two cases of retinal detachment after prolonged observation of the eclipse, one of them in a myopic eye. This detachment has also been observed, without any eclipse, by prolonged exposure to the sun's rays. This last case was observed by Dr. Santos Fernandez and myself in a gentleman who was a spectator at the Willard-Johnson fight in Havana years ago, and who, after being three or four hours exposed to the direct solar rays, had a total detachment of the retina in one eye.

MR. J. GRAY CLEGG (Manchester, England): I would like to emphasize the wisdom of what Dr. Mackay suggests about a list of subjects to be discussed. I was personally unaware until I arrived here just what was coming up. I shall only mention a case which is referred to in a letter that I received two days ago. A little boy got some glass in one eye and as a result had sympathetic disease. He had recovered from the sympathetic disease with perfect acuity, but had another attack of iridocyclitis six months ago. During my absence, a fortnight ago, he looked at an eclipse of the sun, and a rather severe recrudescence has resulted.

ORBITAL MARSUPIALIZATION AND SUPERIORITY OF ORGANIC GRAFTS OF DEAD TISSUE IN ESTABLISHING A MOBILE STUMP

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Since the year 1885, in which Mules proposed enclosing a glass sphere in the sclerotic in order to create a mobile stump, numerous authors have repeated the operation. The question was also discussed in many scientific meetings, and particularly in 1900 at the International Congress of Paris. It would seem that it might be treated anew in order to determine the results obtained by clinical practice. Moreover, during the last few years experimental researches on grafts of dead tissue have brought fresh data by which ophthalmologists should profit in their choice of the substance to be inserted in the funnel of the ocular muscles.

Though making no pretension to writing the history of the question, we think that it is necessary to recall the successive stages followed by the different operators. Mules, in 1884, had at the same time as v. Graefe, proposed the amputation of the anterior segment and the evisceration of the eyeball in all cases in which total extirpation could be avoided. In 1885 he proposed enclosing in the sclerotic a glass ball; in the following years, Brudnell Carter, A. Frost, Grimsdale, Lang, Kroll and Cross, Bickerton, Swanzy in England, L. W. Fox and Orax, Reeve, de Schweinitz, and Buller in America, experimented with the new process. The enclosure of a glass sphere in the Tenon capsule was also tried; later on numerous substances were proposed—rubber (Pick), celluloid (Lang), paraffin wax (Hepburn), asbestos (Cläiborne), the pith of the elder tree (Elschnig), etc. But the preference of the authors was for precious metals. Kuhnt (1887) had enclosed silver spheres in the sclerotic, but two years later Wagenmann had occasion to study some of his stumps and came to the conclusion that dangerous inflammatory reactions might be produced. Operators then returned to spheres in platinum and particularly in gold. We know that many American ophthalmologists have made use of this substance and that certain of their patients have preserved a mobile stump for many years.

Today, twenty-six years after Mules's communication, we are able to measure the ground covered. To judge by the results obtained and the opinions published, it seems that many ophthalmologists are agreed to attempt to make a mobile stump in all cases where there is no orbital infection. Some are in favor of evisceration with inclusion of a glass or gold ball; others, fearing sympathetic ophthalmia, prefer extirpation and planting a sphere in the Tenon capsule. These two methods constitute, in fact, a part of the foundations on which rests the problem to be solved. The other part is constituted by the choice of the substance to be enclosed.

If we are to judge by the number of ill-assorted substances which have been employed, this second part of the problem is not the least important. Many substances have not given satisfaction, others are heavy, others are rare and costly. Those which have been employed most often are glass and gold, as being the ones producing the minimum of painful or inflammatory reactions. As a corollary, it is patients operated in this way who have most frequently been presented in scientific societies.

However, for an operation not to have come into universal use, in

spite of the examples of wearers of gold or glass spheres who had been operated ten or fifteen years previously, it must have aroused some hesitation and distrust. Many hold that we have not yet emerged from the experimental stage and leaving to others the onus of experimenting, reserve their opinion until they shall be convinced of the excellence and innocuousness of a method.

Indeed, in spite of a few cases remarkable for their duration and the esthetic effect obtained, it must be confessed that the inclusion of a gold or glass sphere does not always succeed. Precocious or tardy expulsion may take place, when the operation has succeeded and after a lapse of several weeks; it is therefore impossible to assert that the mobile stump will be permanent.

ORGANIC AND INORGANIC SUBSTANCES

The expulsion of a gold or glass sphere may take place shortly after the operation, when the asepsis has not been perfect or when an accident occurs, such as a hematoma which distends the tissues and brings about rupture of the sutures. But this expulsion can also take place several years later as if there had been a sort of wearing out of the conjunctiva which covers the sphere. This tissue gradually becomes thinner, a small orifice appears which grows progressively and one day the sphere escapes from the cavity without there having been properly speaking any suppuration. Such an accident may supervene just as well with other inorganic substances such as ebonite, galite, celluloid, asbestos, agar-agar, etc.

As for paraffin wax, injected under pressure or fashioned into a ball (Broëkart, Camann, M. Ramsay, Spratt), it likewise has caused many disappointments. Generally speaking, its point of fusion is too low. When it is 75° C. (like that used by Hertel), its superiority over ebonite lies only in the facility with which the operation can give it the desired shape.

It was certainly for the purpose of obtaining a permanent stump that recourse was had no longer to an inorganic substance but to living tissue. Baraquez, Velez, Bartels, Marx, Lauber, recommended the use of fatty tissue, usually taken from the abdomen of the patient and enclosed either in the sclerotic or in the Tenon capsule. This method has been repeated sufficiently often these last few years for it to be possible to judge of its value. The results of recent operations are good, those of long standing are disappointing. As Marx has noted in his histologic examinations, the graft is re-

absorbed and the stump becomes of insignificant volume. In 1912 Sattler, in 1914 Carlotti and Bailleul tried successfully on man the graft of living costal cartilage, but the long-standing results of their transplantations have remained unknown. In 1915, I in my turn tried this method on men wounded in the war, and I was able to convince myself that it offered many serious inconveniences. First of all must be placed the extreme sensitiveness of this tissue to infection and in consequence of this frequent premature eliminations. Secondly, living cartilaginous tissue, like fatty tissue, is absorbed in a few months. This disappearance is the more rapid as the thin and flat human costal fragment is far from being sufficiently voluminous to constitute (even in the beginning) a good stump.

We shall merely make a passing mention of the living heteroplastic grafts of dog's or cat's eyes (Lagrange, Rohmer, Boiadjeff) whose atrophic evolution is similar and we shall now speak of a very interesting category of substances: we refer to dead organic transplantations.

Belt, shortly after Mules's communication, had the idea of implanting in the Tenon capsule a piece of sponge, following in this a study of Hamilton's which appeared in 1881. Hamilton had repeated experiments already made by Stricker (of Vienna) consisting in keeping under supervision the evolution of aseptic sponges introduced into the peritoneum of laboratory animals. He had noted that the framework of the sponge served as guides for the vessels to fresh connective tissue and that at the end of a certain time the organism completely incorporated this exogenous tissue. Belt's experiment was renewed by Trousseau and by Valude, these operations were not successful: elimination took place in the first few days. Much more favorable were the attempts of Schmidt (1910) who made use of globes of bone sterilized by steam. There was no elimination and the stump kept its primitive dimensions. De Wecker and Kuhnt also obtained favorable results with sterilized bone; and if this method has not become more universal, it is probably due to the difficulty of finding spheres cut to the necessary dimensions (fifteen to twenty millimeters in diameter) and ready for use.

In 1915, having had disappointments in attempting to graft living costal cartilage in the Tenon cavity, I had the idea of practising on wounded soldiers inclusions of calf cartilage fixed in formalin of 20% for a week, and then carefully washed. My intention was to proceed by two stages. First to make an inclusion of dead cartilage, pre-

liminary operation, whose object was to avoid elimination of the living cartilage which is so sensitive to infection. I desired in this way to get over the dangerous period and three months later to remove the temporary inclusion and graft in its place costal cartilage, which according to my idea, was to form a permanent stump.

The insertions of formalinized cartilage were successful, but my astonishment was great on finding that after several trials the living transplant rapidly diminished in volume whereas the first had not varied. I therefore modified my mode of operation and systematically employed in all cases of extirpation inclusions of formalinized cartilage. In 1917 I presented to the Paris Ophthalmological Society a number of patients operated since twelve months. In 1918 I presented some more, but I published no monograph on the subject, wishing to submit the process to the proof of time. Today after six years' practice, and an experience of sixty cases, I can speak with a full knowledge of the subject. In the meanwhile researches on grafts have made clear certain obscure points. They have brought to light certain biologic conditions which it is indispensable to know.

TRANSPLANTS OF DEAD TISSUE

For a very long time it was believed that only living tissue could be grafted, that is to say once transplanted they were accepted by their host's organism, and that they continued to live on their own account. All that was known was that the chances of success were slight, and that even under the most favorable conditions (autoplasty) there were for grafts without pedicle about 40% of failures. In the years following the great war, a considerable number of prosthetic operations were attempted on the limbs, radiographic examinations were multiplied, experimental studies were carried out and it was discovered that the vascular, osseous, tendinous or nervous grafts which appeared to be really living, in reality *died* a short time after their transplantation. But, and it is of capital importance, it was remarked that simultaneously *their carcass served as a framework for a young tissue*, the newly formed cells of which took the place of those of the transplant. This phenomenon occurs on condition that there be contact between the graft and the tissue of similar nature of the organism of the host.

Here is therefore a new conception of living grafts which revolutionizes our old ideas and explains the bad results obtained by transplantation into the orbit of living cartilage. It is a sort of biologic

law to which the only exceptions appear to be the cutaneous epithelium and the corneal epithelium.

But if all living tissue (nerve, bone, cartilage, tendon) dies, it must be remembered that its presence suffices to provoke the appearance of young cells of the same nature, and it comes to mind immediately that a transplantation of dead tissue ought to provoke the same phenomenon. Experiments have shown that this is so, and that nerves, tendon or bone killed by alcohol or formalin, fulfilled quite as well and even better their rôle as framework for the new tissue. These facts explain Hamilton's remark, which we have previously mentioned, and enable us to understand how an aseptic sponge can be invaded by connective tissue. For the same reason we can no longer feel astonished at the good results obtained by Schmidt and by Kuhnt, in making use of bone to execute Mules's operation.

Professor Nageotte (Paris) has likewise shown (1920) how the tissues of the host act differently with an inorganic transplant, such as glass, gold, ebonite, etc., and with a transplant of organic tissue killed by histologic fixation.

A disk of glass, silver or of rubber, inserted under the skin of a rabbit's ear, causes no reaction of the tissue: it encysts and the membrane of this cyst is itself insignificant. If the pocket be opened, the foreign body will be found there in a state of liberty. The result is quite different if we insert, in the same place, a disk of bone or cartilage previously fixed in alcohol or formalin, that is to say, a disk of substance organized but deprived of life. *Union almost immediately takes place by continuity of substance* between the inserted piece and the connective tissue of the host. In the case of cartilage this adherence takes place through the perichondrium. In the case of dead bone an analogous phenomenon occurs. The natural surfaces adhere to connective substance of the host, but neither in the case of cartilage nor of bone do the surfaces of section join with it.

Independently of this union between dead and living tissue, a phenomenon which already differentiates entirely the inclusions of organic or inorganic substances, there appears later a new factor of great interest. When the transplants are osseous or cartilaginous, there *appear at the points of contact of the dead tissue osseous nodules* or again cartilaginous nuclei produced by the connective tissue of the host. It follows that, at the end of a certain period a fresh skeleton piece is formed which envelops the transplant. The latter enveloped

in a living sheath will persist unless young living tissue substitute itself for it progressively.

From these facts, we now know that when introduced into the organism organized substances, even when they are deprived of cells and unable to be re-inhabited, take up once more a rôle in the life of the tissues. We have at the same time the explanation, not only of the perfect toleration of dead cartilaginous or osseous fragments transplanted into the orbit, but also of their persistence without diminution in volume.

CARTILAGE FIXED IN FORMALIN

I chose cartilage in preference to bone because it can be cut as easily as a potato. The sternal piece of large-sized calves is, however, the sole material voluminous enough to allow the cutting out of fragments of twenty to twenty-five millimeters in thickness and of the length required. One should be very careful *not to remove the perichondrium*, as this membrane will be the point of union between the transplant and the tissues of the host. This fresh cartilage is fixed during eight to ten days in a solution of formalin, 20%. The formalin is then decanted and replaced by sterilized water. This substance is ready for use two weeks later when it has had time to disgorge the fixing product. This formalin will permeate the water and in this weak antiseptic solution, the fragments will keep indefinitely, forming a reserve on which one can draw according to requirements.

The mode of operation I followed is very simple. After extirpation, the fragment is inserted into the muscular funnel. It is necessary that hemostasis should be complete. The edges of the muscular plane are seized with small clips the vertical traction of which keeps the pocket open. The fragment, which should be as large as possible, is then pushed into the bottom, and the pocket is very carefully sutured with catgut. Over this muscular layer the conjunctiva must be closed with U-shaped stitches so as to make sure that the edges must meet perfectly. I terminate the operation by a temporary blepharorrhaphia which I suppress on the third day. This sealing of the eyelids is a precautionary measure in case of secondary hemorrhage which would distend the pocket. Its effect is to prevent the chemosis which often results from the rubbing of the eyelashes on the conjunctiva.

The patient is then furnished with a binocular dressing for three days in order to immobilize the ocular muscles.

Since 1917 I have always followed this same technic after having assured myself of its perfect efficacy.

I consider as essential:

1. The suturing of the muscles and of the conjunctiva in two planes with separate stitches.
2. Complete hemostasis.
3. The inclusion of a voluminous cartilage from which the perichondrium must not have been scraped off.
4. A binocular dressing for three days.

All those who may try the implantation of formalinized cartilage by this method, will perceive the ease with which the organism accepts this dead tissue. Except in the case of panophthalmia or of orbital infection, recourse may be had to it and I have had numerous successes after extirpation for laceration of the eyeball by projectiles of war. Elimination in the first days will only take place if there is infection, serious hematoma and if the cartilage has been deprived of all its perichondrium. But if there is no elimination in the first fortnight, the transplant may be considered as permanent, for in my cases I have never had a tardy elimination.

Dead cartilage offers over living cartilage the great advantage of being able to furnish voluminous fragments. Whatever its origin may be, it is nevertheless always made up of pieces rather long and thick. The cross section is at the two extremities whereas the other faces are covered with the perichondrium. When inserting the transplant into the cavity, I proceed so that the sides covered with this connective sheath may be in contact with the rectus internus and externus muscles, so that the adherence may take place principally with the muscles which control the horizontal movements.

MARSUPIALIZATION AND TRANSPLANTATION

I have just spoken of the transplantation of formalinized cartilages at the moment of extirpation. This operation is, however, still possible in cases of long-standing extirpation where the patients are not satisfied with their immovable stump. The only conditions required are: a clean conjunctival cavity and behind a small mobile cluster formed of retracted muscles. Briefly, extirpation must have been practised in a proper manner.

The problem consists in creating in the midst of the fibromuscular

mass at the bottom of the orbit a pocket (marsupialization), in the middle of which will be inserted the fragment of cartilage. This result is obtained by incising and then freeing the conjunctiva from its deep attachments.

The fibromuscular mass is then seized by its center, an opening is made in it, and this is enlarged by means of a forceps. The edges are drawn forward and the cartilaginous transplant is pushed in. The muscles are then sutured with catgut, the conjunctiva with silk and we terminate by a blepharorrhaphia as in an ordinary operation.

In December, 1921, I presented to the Ophthalmological Society of Paris an officer who had been obliged to undergo the operation of extirpation during the war. Two years later, immediately after the armistice, he came to see me, complained of his immovable stump, and asked me to remedy it. He was operated in February, 1919. The consequences of the operation were very simple, and for three years he has been wearing a movable stump which has not diminished in volume. The transplant, which is very large, raises the eyelid well, and avoids that ugly fold which cannot be prevented in ordinary enucleation.

CONCLUSION

The superiority of inclusions of dead organic tissues to living tissues is established by clinical practice. When the inclusions are assimilated by the organism, they do not diminish in volume. The fundamental difference between inclusions of inert, unorganized tissues, such as glass or gold, and dead organic tissues, such as bone or cartilage, lies in the fact that the former are foreign bodies tolerated and encysted, whereas the latter are adopted and young osseous cells are seen to appear in them. The former may be expelled one day, whereas the latter, once admitted, are permanent.

These inclusions may be made at the same time as the extirpation, but they can also be effected on extirpations of long standing, provided that this extirpation has been properly made. It is in this case necessary to create a pocket in the middle of what remains of the muscles, but the results obtained are often excellent and comparable to the best stumps consecutive to amputation of the anterior segment.

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DISCUSSION

DR. J. N. ROY (Montreal, Canada): J'ai été fort heureux d'entendre la très intéressante communication de monsieur le Docteur Magitot et d'après ce qu'il nous a dit, je viens à la conclusion que les greffes adipeuses et cartilagineuses agissent différemment en autant qu'elles sont placées dans la cavité oculaire ou dans un endroit quelconque de la face ou du crâne. En effet, j'ai eu l'occasion pendant et après la guerre de pratiquer un nombre considérable de greffes adipeuses, cartilagineuses et osseuses pour réparer des lésions de la figure, des maxillaires et du crâne, et c'est le résultat de mon expérience personnelle, comparé à ce que vient de nous exposer le conférencier, que je désire vous soumettre très rapidement.

D'après M. Magitot, la graisse placée dans la cavité oculaire ne rendrait

aucun service, puisqu'elle se résorbe en totalité dans un espace de temps relativement court. Je serais curieux de savoir si, après l'avoir expérimenté, il a constaté cette transformation partielle du tissu adipeux en liquide huileux que l'on observe quelquefois, et qui s'écoule ensuite de l'endroit où le greffon a été placé. Mise dans la figure ou le cou, pour réparer esthétiquement une dépression locale, la graisse donne d'excellents résultats, surtout si on a eu la précaution d'en mettre une quantité plus considérable que la cavité à combler, et après l'opération, d'appliquer un bandage compressif pour la faire durcir, tout en diminuant son volume à volonté. Ce greffon s'adapte très bien aux tissus environnants qui ont la même consistance que lui, et si dans ce cas, il se résorbe très peu, comparativement à ce que l'on rencontre dans l'oeil où la graisse disparaît totalement, ce phénomène est probablement dû au fait que placée dans la cavité fibreuse de la coque oculaire, cette graisse, d'éléments différents avec ceux qui l'entourent, perd sa vitalité et se résorbe plus ou moins rapidement.

J'ai eu aussi l'occasion de me servir de morceaux d'éponge stérilisée pour remplir des dépressions à la figure. Je dois dire que dans tous les cas je n'ai eu que des succès. Ce corps étranger, en contact avec le tissu adipeux, a pour propriété d'user les cellules, de les ramolir, et de les transformer en une substance huileuse aseptique—contrôlée par le microscope,—et ce liquide s'échappe à l'extérieur jusqu'à ce que le morceau d'éponge soit à son tour éliminée.

Monsieur Magitot nous a dit que la greffe cartilagineuse humaine, placée dans la coque oculaire, se résorbe toujours, tandis que les zoogreffes—un morceau de cartilage de veau par exemple, gardé pendant une dizaine de jours dans une solution de formaline—ne se résorbe pas. Ce fait est encore excessivement intéressant, puisqu'il est reconnu que les greffons de cartilage animal employés pour réparer une lésion du crâne, du sinus frontal, de la paroi orbitaire, de l'os malaire, du nez et du maxillaire inférieur se résorbent toujours, tandis que les greffons pris sur un être humain ne disparaissent jamais, en autant que l'opération est faite aseptiquement. D'ailleurs les remarquables travaux de Morestin sur ce sujet sont connus de vous tous, j'en suis sûr, et ce très habile chirurgien greffait même des morceaux de cartilage pris sur une autre personne. Les examens microscopiques ont prouvé que la greffe animale servant à réparer un traumatisme du crâne ou de la face, se transforme en un tissu fibreux, et qu'elle se résorbe ensuite dans un espace de temps plus ou moins long.

Aussi étant donné la manière tout-à-fait différente dont se comportent les greffons de cartilage humain et animal, je suis porté à croire que le fait de garder pendant quelques jours ces morceaux de zoogreffes dans une solution de formaline, a pour propriété de leur donner plus de stabilité, et de les empêcher ensuite de se résorber; et en terminant, je serais très heureux de connaître l'opinion du conférencier sur cette hypothèse.

DR. A. MAGITOT (closing): La question que me pose le Dr. J. N. Roy concerne deux points: la greffe vivante, la greffe morte.

En matière de greffe vivante il faut faire trois parts: l'hétéroplastie c'est à dire lorsque la greffe est prélevée sur un individu d'espece differente, l'hom-

plastie lorsqu'elle est prise sur un individu de même espèce et l'autoplastie sur l'individu lui même.

Depuis longtemps il est bien connu que l'hétéroplastie vivante est décevante et je prie mon aimable interlocuteur de bien vouloir à ce propos se reporter à mon premier mémoire sur les greffes cornéennes qui a paru en 1911 dans les Annales d'Oculistique.

En ce qui concerne l'homoplastie, ses chances de succes ont paru meilleures. En réalité, les faits cliniques et expérimentaux sont assez nombreux pour qu'on puisse juger de leur valeur, et je conseille au Dr. J. N. Roy qui évoque le souvenir de Morestin de bien vouloir lire les discussions qui ont en lieu à la Société de Chirurgie de Paris à propos des greffes osseuses. De même je lui recommande la lecture des bulletins de la Société de Biologie et les notes si intéressantes du Prof. Nageotte. De tout cela, il ressort que si j'avance que les greffes vivantes meurent au bout de peu de temps et se résorbent, ce n'est pas seulement une vue personnelle, mais le résultat auxquels sont parvenus d'éminents expérimentateurs. Il faut du reste bien remarquer que cette greffe qui ne survit pas est très utile par sa présence puisqu'elle sert de trame à un tissu nouveau. Le résultat cherché est donc souvent atteint.

Je passe maintenant à la greffe de tissu adipeux. De nombreux confrères y ont eu recours pour l'orbite. Les résultats immédiats sont excellents les résultats éloignés sont détestables. Les cellules adipeuses qui à l'état vivant sont semi-liquides, laissent échapper leur contenu huileux et ne sont pas pénétrée par un tissu nouveau. La charpente est insuffisante pour exciter la production d'une masse conjonctive jeune et Marx (cité par moi) a parfaitement mis cette question au point.

La greffe de tissu organique mort ne peut être considérée comme homo-ou hétéroplastique. Il s'agit là d'une masse *impersonnelle*, osseuse ou cartilagineuse qui par la *présence du tissu conjonctif qui la recouvre excite la production de cellules mésodermiques* qui finissent par prendre le type osseux. Voilà pourquoi des transplantations de tissu fixé histologiquement réussissent. Peu importe que ce soit du cartilage ou de l'os de veau. C'est le tissu conjonctif qui importe. Ou aurait sans doute un résultat comparable avec une grosse masse tendineuse.

Si le Dr. J. N. Roy veut bien essayer pour les réparations faciales le cartilage formolé, ou fixé dans l'alcool, il est probable qu'il aura de bons résultats. Je l'ai fait souvent pendant la guerre lorsque je dirigeais le service ophtalmologique du groupe des armées de l'Est; je l'ai fait depuis. Mais pour réussir, il faut plus encore que pour l'orbite, que la plaie faciale soit parfaitement aseptique, et que l'on ait attendu pour intervenir suffisamment de temps après la cicatrisation afin de ne pas réveiller un processus inflammatoire encore récent. J'ai ainsi refait des arcades sourcillières, des pommettes, des rebords orbitaires inférieurs. Nul doute qu'on puisse utiliser le même matériel pour le nez.

LA PERITOMIA EN LAS ULCERAS CRÓNICAS VASCULARES DE LA CORNEA

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En Tratados clásicos y Enciclopedias oftalmológicas se habla generalmente solo de la Peritomia al tratar del pánus de la cornea. Los autores se refieren por lo común á esta intervención quirúrgica al estudiar el tratamiento de las Keratitis vasculares consecutivas á las oftalmias escrofulosas ó al Tracoma. Las primeras producirían con más frecuencia el pánus superficial ó pánus tenuis; el segundo el pánus crasus llamado tambien, tracomatoso ó sarcomatoso, en cuya genesis toma parte con frecuencia la triquiasis el entropión y el ectropión.

En él excelente tratado del Prof. de Schweinitz se recomienda en la Keratitis panosa tocar con el galvano cauterio el lado convexo del creciente vascular ó hacer una libre peritomía.

En la última edición inglesa de la obra del Prof. Fuchs el Prof. Duane traductor, hace la anotación interesante que la peritomía se práctica para curar un grueso pánus y algunas veces en otras condiciones como la episcleritis, herpes, acne rosacea.

Verhoeff—dice Darier en sus Tratados de Therapeutica ocular—que considerando la acnea corneal de naturaleza neuro-trófica, práctica en esta enfermedad una peritomía despegando la conjutiva y cubriendo con ella las lesiones corneales.

En la Enciclopedia Francesa de Oftalmología el Dr. Valude habla solamente de la Peritomia al referirse al pánus corneal.

En el bello libro de cirugía del Dr. Tersón se afirma que la peritomía es un medio precioso en muchos casos para los pánus de toda naturaleza y para las manchas vascularizadas de la cornea. Se agrega: “aciertas úlceras pueden también beneficiar.”

En el reciente tratado de cirugía ocular del Dr. Terien, asi como en la obra tan completa de oftalmología del Prof. Axenfeld, no se habla de la peritomía sino para combatir el panus tracomatoso.

Como se vé por lo anteriormente expuesto, para no citar sino

algunas solamente de las obras más importantes, no se ha hecho un estudio metódico, especial, técnico, de las indicaciones de la peritomía en las úlceras crónicas vasculares de la cornea, cual creemos nosotros, lo merecería por su importancia.

Nuestra diaria experiencia nos ha enseñado todo lo frecuente que es encontrar enfermos portadores no de panus crasus ó tenuis; no de esas corneas enrojecidas y carnosas consecutivas á la oftalmía tracomatosa ó escrofulosa, en que el diagnóstico se impone con la necesidad de una evidencia física; ni siquiera se trata de un pequeño grupo de úlceras localizadas en algún sector corneal vascularizadas más ó menos intensamente, no. Se trata de una, dos, ó tres úlceras pequeñas, de origen variable, transparentes, ú opacas, solas ó acompañadas de algunas manchas cicatrizales; úlceras que son el punto de terminación de algunos vasos, bien visibles muchas veces sólo con la ayuda de la lente, insignificante al parecer, crónicas y rebeldes á todos, los tratamientos habituales. Muchos de estos pacientes han recorrido clinics y gabinetes oftalmológicos donde han sido tratados durante muchos meses por toda la gama de excitantes: calomel oxide amarillo de Hg, dionina, fomentos calientes, masajes eléctricos, etc., etc., sin obtener mejoría alguna. Una peritomía ignea ha terminado en 8 ó 15 días en una completa curación de los pacientes. Estas úlceras no producen generalmente grandes molestias; algo de lacrimoso, algunos pequeños dolores, y son casi compatibles con la vida ordinaria. Los enfermos, sobre todo cuando las úlceras están acompañadas de opacidades de la cornea, preocupados más que por otra causa por la disminución de la agudeza visual, consultan frecuentemente en este sentido al oculista. No siempre estas úlceras son resultado de oftalmias tracematosas ó impetiginosas, se observan como consecuencias de herpes corneales, keratitis tuberculosas, como el caso que relatamos después.

¿Cual deberá ser en estos casos el criterio del oftalmologo? Es principio de Patologia Gral. que los vasos son los principales encargados de llevar los materiales de reparación a los tejidos, y parecería paradójico interrumpir los elementos vasculares para la cicatrización de una úlcera corneal. Que elementos informarán entónces el juicio del especialista para saber el momento en que los vasos, en lugar de conducir elementos útiles á la reparación de una úlcera en la cornea, llevan las celdillas emigratrices de la opacificación y de la esclerosis, que profundizando la membrana de Bowman harán indeleble la lesión, como lo han demostrado los bellos estudios de Iwanoff?

Varios elementos deberán á nuestro juicio ilustrar el criterio clínico del oftalmologista. Desde luego la antigüedad de la lesión. Úlceras crónicas rebeldes á todo tratamiento, deberían hacer pensar siempre en la posibilidad de úlceras mantenidas por vasos anormales de la cornea. Nunca el oculista deberá despreciar en estos casos examinar á la lente de una manera cuidadosa la membrana trasparente. El factor causal se presenta bajo la forma de vasos superficiales, tortuosos, varicosos, poco sensibles á la acción de la adrenalina. Son estas úlceras corneales a las que se refiere Darier sin duda en su Tratado de Terapéutica, con el caracter de no teñirse con la florescina.

El tratamiento en estos casos por medio de todos los excitantes y aun el recubrimiento conjuntival, es inútil. La peritomía es la terapéutica heroica que salvará ojos destinados, de otra suerte, á la pérdida de la visión por opacidad y esclerosis corneal.

La peritomía es una de las operaciones más antiguas de la oftalmología. Aunque se atribuye á Furnari como el inventor á mediados del pasado siglo, es mucho más antigua, pues según Tersón se encuentra ya en Guy de Chauliac, quien la tomó de los árabes.

La operación está constituida por la desinserción conjuntival al derredor de la cornea. Si en lugar de una simple incisión se reseca una lengüeta de tejido conjuntival, se llama periectomía ó sindectomía.

Algunos autores como Panas conceptuan suficiente la simple peritomía y dividen los vasos muchas veces por medio de la punta fina del termo ó galvanó-cauterio, lo que se ha llamado la peritomía ignea.

Nuestra experiencia nos ha enseñado que la peritomía ignea no sólo tiene ventajas por la rapidez de su ejecución, sino que nos parece de especial efecto, para evitar reincidencias, debido a la esclerosis consecutiva á la cauterización.

Esta intervención quirúrgica no sólo produce excelentes resultados para la cicatrización de las úlceras crónicas vasculares de la cornea, sino la mejoría de la opacidades que los acompañan, imposible de obtener con las inyecciones subconjuntivales y demás medios terapéuticos conocidos.

Siendo prolija la descripción de los múltiples casos que en relación con este estudio hemos observado en nuestra práctica, y fuera del propósito de estas líneas, recordaremos muy brevemente los tres últimos, de grande interés clínico.

Obs. 1

H. M. Mujer de 30 años escrofulosa. Padece desde su infancia de los ojos. El ojo izquierdo tiene una serie de pequeñas manchas cicatrizales en la cornea, huellas de antiguas erupciones flictenulares, obstruyendo en parte la pupila y habiendo producido un estigmatismo irregular que compromete algun tanto la visión. OIV = $1/3$. El ojo derecho tiene varias manchas que obstruyen completamente la pupila, dejando libre un sector infero interno de la cornea, y 2 ulceraciones centrales á donde van á terminar algunos vasos delgados, tortuosos, poco visibles, viniendo de la de parte superior del globo.

La agudeza visual de la enferma se reducía á contar los dedos á 2 metros de distancia, teniendo este ojo lacrimoso y adolorido. Tensión normal.

Cuando 1 vimos a esta enferma tenía 9 meses de tratamiento casi continuo con oculistas en varias partes de la Republica, bajo la acción de la pomada amarilla de hg., calomel, dionina, atropina, inyecciones subconjuntivales, tónicos, yodurados, inyecciones intramusculares de hg., etc., sin obtener alivio alguno.

El 10 de Enero de 1920 practicamos una peritomía ignea superior con el galvano-cauterio fino, haciendo la sección profunda de los vasos corneales. Oclusión del ojo que se cura diariamente. A los 15 días no sólo estaban las úlceras cicatrizadas, sino las manchas acompañantes se redujeron y esclarecieron notablemente. Una iridectomía infero-interna practicada á la enferma después, le hizo ascender su agudeza visual á $1/3$.

Obs. 2

V. T. Niño de 12 años de edad linfático, ha padecido desde hace varios años ataques de Kerato conjuntivitis impetiginosa en ambos ojos. El último, que data de siete meses, ha dejado un pequeño grupo de ulceraciones en el sector infero interno de la cornea, vascularizadas y rebeldes á todo tratamiento. Consulta con varios especialistas de la frontera norte de México y de San Antonio, Texas, que lo tratan por medio de tónicos, fiero, arsenicales, y localmente con fomentaciones calientes, pomada amarilla, dionina, etc., sin resultado.

El 10 de Mayo de 1920 le practicamos una peritomía en todo la parte inferior ó interna del globo, con infiltración conjuntival a la novocaina adrenalina. El resultado fué verdaderamente sorprendente, pues á los 10 días se encontraban las úlceras absolutamente cicatrizadas sin haber dejado opacificación alguna.

Obs. 3

M. C. Señora de 50 años de edad. El año de 1918 le atendimos un lupus de la nariz que había invadido la conjuntiva y borde corneal del ojo izquierdo. Le tratamos por inyecciones de tuberculina con excelentes resultados habiendo desaparecido por completo la lesión. Vuelve á enfermarse su ojo izquierdo á principios de 1920 poniéndose el órgano rojo, doloroso y disminuyendo notablemente la visión. Consulta con varios oculistas de la capital, ausente nosotros de ella, á la sazón, que le tratan por medio de fomentos calientes, dionina, argirol, pomadas varias, masaje eléctrico, etc., sin resultado favorable. Volvemos á ver la enferma en Agosto de 1921, y nos encontramos una úlcera central de la cornea de forma redonda y de 2 mils de extensión, invadida por dos ó tres vasos delgados, tortuosos, desprendidos de la parte superior del globo y acompañada por una corona de manchas cicatrizados que ocupaban gran parte de la cornea. La úlcera no se teñía por la florescina. El ojo lacrimoso, sensible á la luz y doloroso, y con la visión perdida. La tensión del órgano era normal. No se había reproducido el antiguo lupus nasal.

Ante la rebeldía de 1 año y medio á todos los tratamientos de aquella lesión ocular, practicamos á la enferma desde luego el 15 de agosto de 1921 una peritomía ignea superior dividiendo á la lente binocular todos los vasos que penetraban en la cornea. Desde el tercer día se comenzó á ver una mejoría notable en el estado local de la enferma y á los 20 días se encontraba la úlcera absolutamente cicatrizada. Las manchas corneales se han esclarecido de tal modo después de la intervención, que la paciente ha alcanzado una agudeza visual de 1/10 y rehusado á una iridectomía óptica que le habíamos propuesto.

Esta breve comunicación podríamos sintetizarla en algunas conclusiones. 1a. El unico tratamiento eficiente, y con frecuencia heróico, de las úlceras crónicas y vascularizadas de la cornea es la Peritomía. 2a. En toda úlcera corneal crónica rebelde a los tratamientos varios usuales, el oculista debiera sistematicamente examinar la cornea á la lente, para definir si no existen vasos mantenedores de la lesión. 3a. La frecuencia de estas ulceraciones, su rebeldía á la Terapéutica ordinariamente, empleada, y las consecuencias funestas que tienen para la función visual, meritan que los tratadistas los consagren una atención más amplia y un sitio más importante en la nosografía, oftalmológica.

DISCUSSION

DR. L. WEBSTER FOX (Philadelphia): Considerable credit is to be given to this author for bringing to the attention of the ophthalmic world the value of this therapeutic measure. Many times in the past have prominent surgeons noted the value of this operation but have failed to impress it sufficiently upon their students and followers, so that it has just so often been allowed to fall into the discard, to be revived from time to time by some progressive surgeon who has had to discover for himself the great benefit to be derived from its employment.

Dr. Alonso adds a note to the indications for its use when he advises the operation in chronic vascular ulcers of the cornea. His comment that it has been referred to mostly in connection with pannus secondary to scrofulous and trachomatous inflammation is accurate, and it is rather unfortunate that the authorities he quotes should be averse to admitting of its larger possibilities, and indeed rather reluctant to acknowledge even its usefulness in that obstinate condition, pannus. The French have given it a much higher status than the Americans, as Dr. Alonso has shown. His personal experience with intractable ulcers is of great interest and shows close attention to minute clinical detail. It is most certainly necessary, as he implies, to bring about resolution in these cases in order to avoid the scarring of the cornea and the consequent disturbance of vision.

It is agreeable to note that Dr. Alonso stresses the importance of good ophthalmic judgment in deciding when the vessels leading to one of these ulcers has ceased to conduct useful elements for the reparation of the ulcerated site, and instead carry migratory cells of opacification and sclerosis. He regards with good cause the age of the ulcer as an index of its pathologic condition. The chronic rebellious ulcer is maintained by normal blood-vessels. It is in this phrase of Dr. Alonso's that the whole story of rebellious ulcers is told. The vessels are abnormal. At no time are blood-vessels normal to the cornea, and these fine red lines which we see in these cases, while they carry blood are not blood-vessels in the histologic sense but are the same capillary loops that we see in all forms of exuberant granulation. These ulcers are in fact exuberant granulations. In any other situation the surgeon would entertain no other treatment than complete eradication. Dr. Alonso prefers what he terms "igneous peritomy," which consists of division of the vessels many times by means of the fine point of the galvano-cautery. The scarring produced by this cauterization is believed to prevent recurrences.

There must be a great merit to this method as his experience coincides with my own. My own preference has been for a clean excision of the conjunctiva surrounding the cornea, and an experience of over a thousand cases in the last twenty years since I first used this method convinces me that my selection of this technic is the correct one. The operation is a modification of that of Furnari and is known in many circles as "peridectomy."

The technic consists first of anesthetization of the conjunctiva with a five per cent. solution of cocain, and the additional installation of a drop or two of adrenalin solution to control slight hemorrhage. A strip of bulbar

conjunctiva from 5 mm. to 15 mm. immediately surrounding the cornea is then excised with sharp scissors. The vessels on the cornea at the limbus are then scarified by means of a sharp curved knife devised by myself but no cauterant is used. Special emphasis must be laid upon the necessity of a clean cut incision of the conjunctiva. Chewing of the conjunctiva with dull or imperfectly set scissors will induce an unnecessary inflammation.

When Furnari brought forth his operation in 1842, trachoma was present in a most virulent type in the French Army and in the French Colonial possessions, and his enormous experience with this disease in Algiers prompted him to the development of a radical technic in the expectation of checking the ravages of the disease. His operation consisted in first making an incision at the outer canthus, after which a broad ring of bulbar conjunctiva was excised extending from the margin of the cornea to within 3 mm. of the line where the conjunctiva is reflected from the globe to the inner surface of the lid. A flap of conjunctiva at the margin of the cornea was allowed to remain to the last in order to fix and rotate the globe at the will of the surgeon during the operation. The conjunctival tissue was then carefully dissected away so as to expose the sclera to view. The vessels of the cornea were then scarified, after which the sclera and ulcerated portions of the cornea were touched with silver nitrate.

The success attending Furnari's cases led other surgeons to practise this operation but with innumerable modifications. One of the early and probably the best, was to discard the cauterizing feature. This is mentioned here in order to call attention to the fact that attractive as the cauterizing may appear, it has always been discarded after any prolonged experience with it. However, it may be that the use of the galvano-cautery may be a little more elegant and give a much better result than the cauteries used in bygone days. In closing the discussion I wish to call the author's attention to my article in the American Annals of Ophthalmology, October, 1903.

DR. S. LEWIS ZIEGLER (Philadelphia): I want to endorse what Dr. Fox has said in regard to the advantages of peridectomy over peritomy, or peridotomy as it should be designated. The operation of Furnari is successful in cutting off the vascular supply in these cases. I recall, however, that in addition to this Dr. Fox, about thirty-five years ago, used Burow's operation to release the tension of the lid.

The two points in these cases of vascular ulcer are—(1) the maceration and (2) the lid tension. I have used galvano-cautery peridectomy extensively, but after 1890 I abandoned it, for the reason that I found I could gain the same result without conjunctival destruction by resorting to rapid dilatation of the tear duct to relieve the maceration and cantholysis to release the lid tension. There is destruction, as a rule, in peridectomy, because if you take off a strip of conjunctiva when it is already atrophic you shorten it and cause xerosis. By stretching the tear duct you relieve the corneal maceration and by the operation of cantholysis you release the tension of the lid, and thereby gain freedom from friction of the cornea. I advocated these measures in a paper on "The Surgery of Trachoma," presented before the American Medical Association in 1898.

There is only one thing that must be observed in this operation. Canthotomy or cantholysis, as Agnew preferred to call it, means the division of the external (superior) canthal ligament. In performing the operation you make a small incision at the external canthus with sharp-pointed scissors, then with forceps you grasp the border of the superior lid at the point of incision, pass in your scissors and feel for the canthal ligament just as you would feel for the optic nerve when you enucleate. As soon as you feel the ligament, open your scissors and divide it, and the corner of the lid will jump up about a centimeter. If you do not get that freedom of the upper lid you have not divided the ligament.

DR. ALONSO (closing): To estimo en alto grado que los Senores profesores que han hecho uso de la palabra á propósito de mi trabajo esten en el fondo de acuerdo conmigo; muy especialmente celebro y estimo las frases del Dr. Fox, que le ha merecido elogios mi actuación en esta asamblea al traer á su seno la question del tratamiento de las úlceras crónicas y vasculariza das de la cornea.

El asunto esencial es que no hay que con fundio el tratamiento quirúrgico del Panus corneal en el cual la peritomia esta recommendada fur les autores con las tridicaciones de esta operacion en las ulceraciones que hacen el objeto de un comunicacion.

Como lo indico ampliamente en mi trabajo estas ulceraciones son muchas veces discretas y mantenidas por vasos quisibles solamente con ayuda de la lente. Todes los tratamientos excitantes conocidos como la dioni na las fomentaciones calientes el exido amarillo de hyla canterisaciones la electricidad, el recubrimiento conjuntival mismo, no producen resultado. Nelo con la peritomia se obtienen resultados excelentes y definitivos, ha gran cuestion es hacer el diagnostico ele sus indicaciones acerca de las cuales insisto ampliamente en mi trabajo. Ulceraciones cronicas vascularizadas, rebeldes á les tratamientos ordinarios, no colorandose por la florescina, etc.

TYPES OF PNEUMOCOCCUS FOUND IN CORNEAL ULCERS

ROBERT CARTWRIGHT CHENEY, M.D.

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(From the Massachusetts Charitable Eye and Ear Infirmary)

In 1902 Römer,¹ realizing the inadequacy of the treatment of pneumococcus ulcer of the cornea and the disastrous end result as far as useful vision was concerned, attempted serum treatment in these cases. Using small doses of immunized animal serum, he had promising results in rabbits. In men, however, the results were by no means

¹ Römer, P.: Experimentelle Grundlagen für klinische Versuche einer Serumtherapie des Ulcus cornea serpens nach Untersuchungen über Pneumokokkenimmunität, Arch. Ophth., 1902, liv, 99.

as striking, and in the five cases treated only three were apparently successful. A significant fact is that these three cases were only forty-three, thirty-one, and sixteen years of age, and hence had a much better chance of recovery under any treatment. Indeed the serum treatment has never proved of any clinical value and has never supplanted cauterization or other previously recognized forms of therapy.

In recent years the value of serum treatment in cases of lobar pneumonia has apparently been proved and a very satisfactory summary is given in a monograph from the Rockefeller Institute by Avery, Chickering, Cole, and Dochez.¹ It was found that pneumococci might be divided into four types on the basis of their specific immunity reactions. To quote directly, "pneumococci of types of I, II, and III, which compose about 80 per cent. of all strains encountered in disease, and which represent three apparently fixed types of highly parasitic organisms, each possessing common immunologic characters." Type III is the pneumococcus mucosus. Type IV pneumococcus is a heterogeneous group and is mostly made up of individual strains which are not interrelated and do not have a common agglutination reaction with any single immune serum. In short, it is possible to obtain three stock sera, types I, II, and III, which have specific agglutination reactions for the corresponding types of pneumococci, but as is evident from the preceding statement, there is no type IV serum, and a pneumococcus which does not agglutinate any of the three sera is relegated to the type IV group. In the actual clinical application of these sera, the type I serum in large intravenous doses is seemingly highly effective in lobar pneumonia caused by a type I organism, but of no use in a case caused by a pneumococcus of another type. Type II and III sera as yet have not been proved to be of clinical value.

In view of the above facts it seemed important to ascertain the types of pneumococcus found in corneal ulcers, with the hope of finding an ulcer caused by a type I organism which might advantageously be treated with serum.

Some difficulty was encountered at first in obtaining a satisfactory culture medium as naturally comparatively little material for inoculation is obtainable from a corneal ulcer. Intraperitoneal injection in white mice was not satisfactory, but Avery medium proved to be just what was needed, and I never failed to get a growth from a pneumo-

¹ Monograph of The Rockefeller Institute for Medical Research. No. 7. Acute Lobar Pneumonia, Prevention and Serum Treatment, by Oswald T. Avery, M.D., H. T. Chickering, M.D., Rufus Cole, M.D., and A. R. Dochez, M.D.

coccus ulcer when this medium was employed. It was described by Avery in 1918¹ and is a meat infusion broth three-tenths to five-tenths acid to phenolphthalein. To each 100 c.c. of broth is added 5 c.c. of 20 per cent. sterile glucose solution and 5 c.c. sterile defibrinated blood.

The following was the method of procedure: the eye was carefully cocainized, a speculum inserted, and as much material as possible obtained by curetting the advancing infiltrated edge of the ulcer, which was found to be the only place where pneumococci were plentiful enough to give a satisfactory culture. Great care should be taken not to traumatize the normal corneal tissue, and the ulcer should be cauterized immediately after curetting, as curetting alone tends to make the process somewhat worse. Next, a tube of Avery medium was inoculated with the material so obtained and, after careful shaking, was placed in the incubator for six to ten hours at the end of which time the blood corpuscles had settled at the bottom of the test tube. The supernatant fluid was almost always found to contain a pure growth of pneumococci and from this the type was ascertained by direct microscopic agglutination with the sera obtained from the New York State Board of Health and the solubility of the organisms in bile determined (pneumococci soluble in bile, streptococci not). Römer called attention to the usual occurrence of a pure growth in pneumococcus ulcer of the cornea, obtaining 18 pure cultures in 20 cases. Thus no centrifuging was necessary, and the culture was directly grouped, the reaction being a combination of agglutination and precipitation. Care should be taken, however, not to allow cultures to go over 10 to 12 hours as the organisms in Avery medium form acid which soon kills them.

During a period of over a year at the Massachusetts Charitable Eye and Ear Infirmary cultures were obtained from 12 cases of pneumococcus ulcer of the cornea. These were pure cultures with but one exception, where there was a slight growth of staphylococcus. In eight of these cases the organism was type IV pneumococcus, while in the other four cases it was type III pneumococcus. No type I or type II organisms were found, the percentage being 66.6 per cent. type IV, and 33.3 per cent. type III. From the standpoint of any serum treatment the results were very disappointing, but nevertheless

¹ Avery, O. T.: Determination of Types of Pneumococcus in Lobar Pneumonia. A Rapid Cultural Method. Jour. Amer. Med. Assn., January, 1918, lxx, 17.

interesting when compared with the following statistics taken from Monograph 7 of the Rockefeller Institute:

1. Occurrence of the various types of pneumococci in lobar pneumonia and the mortality.

Occurrence		Mortality	
Type I.....	33.3%	Type I.....	25%
Type II.....	29.3%	Type II.....	32%
Type II (Atypical).....	4.2%	Type III.....	45%
Type III.....	13.0%	Type IV.....	16%
Type IV.....	20.3%		

2. Types of pneumococci found in mouths of normal persons (297).
Present 116 cases. Absent 181.

Type I.....	0.9%
Type II.....	0.0%
Type II (A).....	0.8%
Type II (B).....	5.8%
Type II (X).....	11.6%
Type III.....	28.1%
Type IV.....	52.9%

3. Types of pneumococcus recovered from dust in rooms in which lobar pneumonia had not occurred.

Type I.....	5.5%
Type II.....	0.0%
Type II (A).....	0.0%
Type II (B).....	22.0%
Type II (X).....	16.6%
Type III.....	11.0%
Type IV.....	44.4%

The preceding statistics show that of the types of pneumococci found in the mouths of normal people 28.1 per cent. are type III and 52.9 per cent. are type IV which compare fairly closely with 33.3 per cent. and 66.6 per cent. respectively as found in corneal ulcers. Thus it is to be wondered if the conjunctiva or the tear sac in clinically normal persons does not also contain type III and IV organisms as well as the mouth.

Of the 12 corneal ulcers studied by me 11 were clinically typical serpent ulcers, but one case needs special description: a boy 13 years old with a severe membranous conjunctivitis and a very superficial corneal ulceration which was secondary to the conjunctivitis. Type III pneumococcus was isolated from the conjunctiva. This eye recovered quickly with no impairment of vision.

In lobar pneumonia, type III pneumococcus is by far the most virulent, carrying a 45 per cent. mortality, so naturally there arises the question of prognosis in eye infections as determined by the type of

pneumococcus. In this extremely small series with various methods of treatment, etc., no definite evidence is obtainable, but it does seem significant that, in the 11 cases of *ulcus serpens*, the only two eyes that required enucleation during the patient's stay in the hospital were infected with a type III organism.

Summary: Of the ulcers studied, 66 per cent. were caused by a type IV organism and 33 per cent. by a type III organism.

The failure of serum in pneumococcus ulcers of the cornea is adequately explained by the fact that none of the organisms are of type I.

The present pneumococcus sera obtainable have little, if any, specific action on organisms in the cornea and absolutely no benefit is to be expected from their use.

That the type IV pneumococci found in the cornea belong to one definite sub-group for which there may be a specific serum, is possible but not probable.

In conclusion, I wish to thank Dr. G. S. Derby and Dr. F. H. Verhoeff for their kindness in giving me valuable suggestions and in allowing me to report their cases.

DISCUSSION

DR. GEORGE S. DERBY (Boston, Mass.): This is an important bit of research, because, according to my knowledge, it is the first time pneumococci found in the eye and corneal ulcer have been typed. It was necessary that this should be done. It explains the failure of sera to work.

To those who think there is some virtue in making bacteriologic examination of secretions in the conjunctival sac, especially before cataract operation, the use of Avery media is certainly to be recommended. It is a comparatively simple medium to make up, and it will grow pneumococci nearly every time. Moreover, it grows pneumococci in from six to ten hours, so that you can have your patient come to the hospital the night before, have a culture taken, and have your bacteriologic report early next morning before you are ready to operate.

DR. R. C. CHENEY (closing): I do not think that serum treatment in pneumococcus ulcers of the cornea will ever be of value, even if type I organisms are found. When you consider the avascular character of the cornea, it is hard to conceive how you could influence the process to any great degree by injection of serum into the blood-stream which is conspicuous by its absence at the site of the lesion.

L'ÉTAT VASCULAIRE DES GLAUCOMATEUX (ÉTUDE DE 100 MALADES DE GLAUCOME PRIMITIF)

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Au mois d'octobre de cette année, nous avons envoyé à la Société d'Ophtalmologie de Paris un mémoire sur l'état vasculaire des glaucomateux, mémoire fait en collaboration avec notre élève, le Docteur Cristobal Espildora.

Nous avons réuni au hasard de leur arrivée à notre consultation 75 observations de glaucomes primitifs où les malades étaient étudiés au point de vue général, surtout au point de vue cardio-vasculaire.

Depuis lors, nous avons poursuivi notre étude et ce sont les 25 nouvelles observations réunies, que nous présentons au Congrès International d'Ophtalmologie de Washington de 1922.

Ce nouveau matériel clinique accumulé a confirmé dans les grandes lignes les conclusions de notre premier mémoire.

Nous faisons précéder ces 25 observations d'un résumé, de l'exposition de la matière contenue dans cette première communication faite à la Société française d'Ophtalmologie de Paris.

Dans les 25 dernières observations nous avons étudié plus minutieusement nos malades:

(a) Nous avons insisté davantage sur l'examen des urines.

(b) Nous avons mesuré à plusieurs reprises, en différentes séances, la pression artérielle quand la normalité de celle-ci se prêtait à des doutes.

(c) Nous avons fait la Réactivation de la Réaction de Wassermann (méthode de Millian) quand celle-ci était négative, malgré des antécédents suspects.¹

(d) Nous avons complété notre examen clinique par une amnèse générale, aussi souvent que cela a été possible.

¹ Nous faisons la Réactivation en injectant $\frac{1}{2}$ cg. de cyanure de mercure intraveineux tous les 2 jours; après la 3ème injection, nous refaisons le jour suivant la Réaction de Wassermann.

I. LES ALTERATIONS VASCULAIRES CHEZ LES GLAUCOMATEUX

Qu'avons-nous trouvé sur les 100 premiers malades examinés:

90 malades avec des altérations cardio-vasculaires;

3 id. sans altérations cardio-vasculaires perceptibles, mais avec Réaction de Wassermann positive;

7 id. avec examen cardio-vasculaire et sérologique négatif.

Nous nous empressons de dire que tous les cas ne se présentaient pas sous un type clinique défini, de constatation et de diagnostic faciles—ainsi, chez quelques-uns, l'affection vasculaire aurait passé inaperçue sans la manométrie, la radioscopie, la sphygmographie et l'examen des urines.

Chez la grande majorité, au contraire, la lésion aortique, l'hypertension artérielle ne pouvaient rester méconnues, même a un examen superficiel.

La localisation clinique prédominante a été l'aortite et l'hypertension artérielle. L'aorte est malade 59 fois sur 100 cas, 15 fois l'aortite chronique était accompagnée d'insuffisance valvulaire, 5 fois de rétrécissement aortique pur et 1 fois de rétrécissement et d'insuffisance simultanée. (obs. 18.)

L'aortite s'est souvent présentée silencieuse sans symptômes subjectifs.

L'hypertension artérielle est encore plus fréquente (62 fois sur 100 malades).

Le compromis du rein constatable par un examen d'urine anormal (faible densité et indices d'albumine) n'est pas très fréquent. Mais si nous devons interpréter l'augmentation de la pression systolique comme un signe de sclérose rénale, nous devons admettre que le rein a dû être touché bien plus souvent et qu'une étude plus approfondie de la fonction de la glande nous aurait permis de constater, chez beaucoup de malades, la néphrite chronique ébauchée.

En effet, au commencement de notre étude, la grande majorité des examens d'urine ont été très superficiels et absolument insuffisants.

La discordance entre l'examen rénal et l'examen vasculaire de nos 65 premières observations nous a surpris et un peu déroutés.

Dans les observations postérieures, l'étude de l'élimination urinaire a été plus complète et nous avons trouvé ce qui nous faisait supposer l'état vasculaire anormal: le compromis fréquent du rein. La

néphrite chronique fruste se présente très souvent chez le glaucomateux.

En tout cas, ces 62 malades, ce 62% d'hypertension systolique parle de la grande fréquence de la sclérose vasculaire périphérique chez les glaucomateux.

La pression diastolique ou minima était augmentée chez quelques malades d'une façon manifeste.

La myocardite a été constatée 5 fois.

A noter 5 malades avec lésion mitrale.

Le diabète s'est présenté 3 fois et accompagné d'aortite et d'hypertension artérielle.

L'emphysème et la sclérose pulmonaire n'ont pas été rares.

La tuberculose pulmonaire n'a été observée en période d'activité qu'une seule fois. (obs. 91.)

Les ganglions bronchiaux ont été décélés fréquemment à l'examen radioscopique.

II. ETIOLOGIE DE L'ALTERATION VASCULAIRE GENERALE CHEZ LES GLAUCOMATEUX

Nous avons dit que sur nos 100 malades, 90 présentaient des altérations de l'appareil vasculaire.

De ces 90, 59 avaient l'aorte anormale et 62 de l'hypertension artérielle.

Les auteurs indiquent comme cause des lésions vasculaires chroniques et spécialement des aortites, les infections et intoxications, par exemple la syphilis, l'alcoolisme, la sénilité, la goutte et le paludisme. De ces facteurs, nous pouvons éliminer la goutte et le paludisme, maladies exceptionnelles au Chili, véritables trouvailles cliniques (nous n'en avons rencontré aucun cas parmi nos glaucomateux).

La syphilis et l'alcoolisme sont au contraire d'une extrême fréquence, surtout la première de ces affections.

Il ne serait pas très aventuré de supposer qu'une grande part de l'action de l'intoxication étylique soit due à la syphilis, que l'on trouve souvent chez les antécédents d'un alcoolique.

La sénilité a une influence, semble-t-il, plus secondaire que la syphilis, puisque entre nos 100 glaucomateux, 41 seulement sont entrés dans l'âge sénile, ayant dépassé les 60 ans, et entre ces 41, 11 étaient sûrement et 3 probablement syphilitiques.

Le Glaucome s'est présenté:

De moins de 30 ans.....	1 fois
De 31 á 40 ans.....	2 “
De 41 á 50 ans.....	26 “
De 51 á 60 ans.....	30 “
De 61 á 70 ans.....	31 “
De 71 á 80 ans.....	9 “
De 81 á 90 ans.....	1 “
100 malades: 35 hommes, 65 femmes.	

En tenant compte des deux grands facteurs étiologiques de l'affection de l'appareil vasculaire, la syphilis et la sénilité, de beaucoup les plus importants, nous sommes arrivés á séparer nos malades en 4 groupes:

1. Glaucomateux avec artériopathies syphilitiques que nous désignerons sous l'expression “glaucomateux syphilitiques.” (Page 11.)

2. Glaucomateux non syphilitiques avec lésions artérioscléreuses, que nous désignerons sous l'expression “glaucomateux artérioscléreux.” (Page 28.)

3. Glaucomateux avec affections cardio-vasculaires d'étiologie diverse. (Page 37.)

4. Glaucomateux á examen général négatif. (Page 39.)

TABLEAU GENERAL. GLAUCOMATEUX

	Total	Syphilitiques	Artérioscléreux	?	Négatifs
De moins de 30 ans.....	1	1
De 31 á 40 ans.....	2	1	1
De 41 á 50 ans.....	26	22	..	4	..
De 51 á 60 ans.....	30	20	8	..	2
De 61 á 70 ans.....	31	9	18	..	4
De 71 á 80 ans.....	9	4	5
De 81 á 90 ans.....	1	..	1
	100	57	32	4	7

III. GLAUCOMATEUX SYPHILITQUES (57 Cas)

Des 100 glaucomateux étudiés, 46 étaient sûrement syphilitiques et 11 très suspects, ce qui donne un haut pourcentage de près de 60%.

Le Glaucome s'est présenté chez des syphilitiques:

De moins de 30 ans.....	1 fois
De 31 á 40 ans.....	1 “
De 41 á 50 ans.....	22 “
De 51 á 60 ans.....	20 “
De 61 á 70 ans.....	9 “
De plus de 70 ans.....	4 “
Total.....	57

Sur le total des 100 glaucomateux, 29 n'avaient pas dépassé la cinquantaine et de ces 29, 24 étaient syphilitiques et 5 seulement apparemment étaient libres de l'affection vénérienne.

Nous croyons que cette constatation doit être soulignée parce qu'elle a une certaine portée pratique.

Notre petite statistique dit: le glaucomateux de moins de 50 ans, le glaucomateux jeune, est presque toujours syphilitique.

Il nous semble que ce résultat statistique n'est pas un simple effet du hasard, mais naturellement cette affirmation a besoin, pour acquérir sa véritable valeur, qu'une observation étendue à un plus vaste matériel clinique, la confirme.

Des 57 glaucomateux, considérés par nous comme syphilitiques ou très suspects de syphilis, 32 avaient une Réaction de Wassermann positive, 1 était hérédosyphilitique, 13 avaient des antécédents spécifiques non discutables, 11 seulement sur ces 57 sont des cas probables, très probables, mais non prouvés.

Entre les 32 malades avec Réaction de Wassermann positive: 3 ne présentaient aucun symptôme cardio-vasculaire, 4 seulement une image radioscopique anormale, 2 une insuffisance mitrale (obs. 12 et 84) et 23 de l'hypertension artérielle ou de l'aortite, etc.

Le compromis du myocarde a été observé 5 fois chez les syphilitiques.

Des 18 glaucomateux, considérés comme syphilitiques, mais avec Réaction de Wassermann négative, 9 avaient de l'aortite chronique, compliquée d'insuffisance valvulaire sigmoïdienne, aortite du type syphilitique; 5 de l'aortite et de l'hypertension artérielle avec des antécédents spécifiques non discutables, 1 avait de l'aortite et présentait simultanément de la névrite optique syphilitique à l'oeil de tension normale (obs. 46), 1 de l'aortite et un rétrécissement aortique (obs. 79) 1 de l'aortite et de la néphrite (obs. 81) et 1 de l'hypertension artérielle (obs. 78).

Le diabète a été observé 2 fois. (Obs. 91 et 96.)

Sur le total de 57 malades l'aortite a été signalée 42 fois, avec lésion valvulaire 18 cas; l'hypertension artérielle 37 fois.

Entre ces 57 observations, nous avons rencontré 5 fois le glaucome aigu.

GLAUCOMATEUX
SYPHILITIQUES

- | | |
|---|---|
| { | 1. Glaucomateux syphilitiques avec Réaction de Wassermann positive. |
| | 2. Glaucomateux syphilitiques avec Réaction de Wassermann négative. |
| | 3. Glaucomateux suspects de syphilis. |

1. GLAUCOMATEUX AVEC REACTION DE WASSERMANN POSITIVE.¹—
Les observations contenues dans ce Mémoire apparaissent soulignées.

(a) Sans manifestations vasculaires générales perceptibles; obs. 11, 16, 82.

(b) Avec image radioscopique cardio-aortique anormale; obs. 6, 9, 51.

(c) Avec symptômes vasculaires généraux frustes (légère hypertension artérielle); obs. 8, 25, 93.

(d) Avec insuffisance mitrale et dilatation du coeur; obs. 12.

(d') Avec insuffisance mitrale et hypertension artérielle; obs. 84.

(e) Avec aortite et sphygmogramme avec plateau; obs. 32, 45, 71.

(f) Avec aortite et hypertension artérielle; obs. 39, 54, 92, 98, 100.

(f') Avec aortite, hypertension artérielle et diabète; obs. 96.

(g) Avec aortite et myocardite; obs. 41, 95.

(g') Avec myocardite; obs. 99.

(h) Avec aortite, rétrécissement aortique, hypertension artérielle et insuffisance mytrale; obs. 66, 53.

(k) Avec aortite, insuffisance aortique et hypertension artérielle; obs. 22, 26.

(l) Avec aortite, insuffisance aortique, hypertension artérielle et myocardite; obs. 23.

(m) Avec aortite, artériosclérose et hypertension artérielle; obs. 13, 37, 21, 28.

2. GLAUCOMATEUX SYPHILITIQUES AVEC REACTION DE WASSERMANN NEGATIVE.

(a) Avec aortite du type syphilitique compliquée d'insuffisance valvulaire sigmoïdienne: (I) Sans hypertension artérielle; obs. 50; (II) Avec hypertension artérielle, obs. 20, 30, 34, 52, 57, 86, 87.

(b) Avec aortite du type syphilitique avec insuffisance et rétrécissement aortique et hypertension artérielle; obs. 18.

(c) Avec aortite, sans lésions valvulaires, avec hypertension artérielle et antécédents cliniques de syphilis; obs. 19, 31, 40, 63.

(d) Avec symptômes aortiques et avec une lésion syphilitique en pleine évolution; obs. 46.

3. GLAUCOMATEUX AVEC SYMPTÔMES VASCULAIRES ET SUSPECTS DE SYPHILIS.

(a) Avec symptômes d'artériosclérose périphérique apparus précocement; hypertension artérielle; obs. 14.

¹ Les observations de 1 à 75 forment partie du Mémoire présenté à la Société d'Ophtalmologie de Paris.

- (b) Avec hypertension artérielle; obs. 78.
- (c) Avec symptômes cardio-aortiques et ancienne Irite; obs. 3.
- (d) Avec aortite et néphrite chronique; obs. 87.
- (e) Avec aortite et hypertension artérielle à 50 ans; obs. 4.
- (f) Avec aortite, hypertension artérielle et diabète; obs. 91.
- (g) Avec aortite et rétrécissement aortique; obs. 79.
- (h) Avec aortite et insuffisance aortique; obs. 42.
- (i) Avec aortite, insuffisance aortique, hypertension artérielle et myocardite; obs. 48.
- (k) Avec aortite, insuffisance aortique, hypertension artérielle; obs. 56.
- (l) Avec hypertension artérielle, néphrite chronique et symptômes aortiques à 44 ans; obs. 55.

I. OBSERVATIONS DE GLAUCOMATEUX SYPHILITIKES

1. AVEC REACTION DE WASSERMANN POSITIVE.—(a) *Sans Symptômes Cardiovasculaires Perceptibles*. Obs. 82 (No. 5661) (No. d'ordre de la clinique).

Rosa A., femme, 55 ans. Diagn. ophtalm.: O. D., glaucome absolu; O. G., glaucome chronique. Diagn. clinique: Syphilis.

Examen oculaire (Juillet, 1921); O. D., cornée opacifiée dans son $\frac{1}{4}$ inférieur; chambre antérieure aplatie; pupille mydriatique immobile; iris atrophique; opacité totale du cristallin. Vision = 0. Tension + 2. O. G., chambre antérieure aplatie; pupille mydriatique moyenne, sans réactions; excavation glaucomateuse profonde, mais rosée. Vision, perception lumineuse. Tension + 2.

Examen clinique: Pointe du coeur au 5ème espace intercostal, ligne mammaire; 2ème bruit aortique renforcé. Pression artérielle (Pachon): Mx. 15; Mn. 7. Radioscopie: Coeur légèrement augmenté de volume—Aorte normale. Urine: Densité 1016; indices d'albumine; peu d'indican et traces d'urobiline. Urée 7.80 %; chlorures 9.0%; au microscope: peu de globules blancs, de cellules épithéliales polygonales et rondes. Sang: Urée 0.20%. Réaction de Wassermann positive.

(b) *Avec Symptômes Cardiovasculaires Frustes*. Obs. 93 (No. 5754).

Fidelisa J., 48 ans, femme. Diagn. ophtalm.: O. D., glaucome aigu; O. G., idem. Diagn. clinique: Syphilis.

Examen oculaire (Août, 1921): O. D., injection bulbaire, cornée transparente, normale, pupille en mydriase moyenne, immobile; fond pas examinable. Vision, O. D., compte les doigts à 40 cm. Tension, Schiötz 1/10 = 70 mm. O. G., injection bulbaire, cornée de surface chagrinée;

pupille mydriatique, immobile, fond pas examinable. Vision, mouvements de la main à 0 m. 50. Tension, Schiötz 1/10 = 70 mm.

8 Août: Iridectomie antiglaucomateuse O. D. en bonnes conditions sous anesthésie locale.

18 Août: Vision, O. D. 5/30. Tension = 1 ou—1. Iridectomie antiglaucomateuse O. G. sans incidents, sous anesthésie locale (Duverger).

8 Sept.: Vision, O. D. 5/15; O. G. 5/40. Tension, O. D. 8/7.5 = 16 mm. (Schiötz); O. G. 12/7.5 = 9 mm.

22 Sept.: Vision, O. D. 5/10; O. G. 5/30. Tension, Schiötz, O.D. 8/7.5 = 18 mm.; O. G. 6/7.5 = 23 mm.

Examen clinique: Renforcement du 2ème bruit aortique. Pression artérielle (5 Août) Pachon, Mx. 16.5 Mn. 8; pression artérielle (6 Sept.) Pachon, Mx. 16.5 Mn. 10.5; pression artérielle (22 Sept.) Pachon, Mx. 17 mm. Mn. 11 mm. Radioscopie thoracique négative. Réaction de Wassermann: positive. Urine: examen négatif. (Densité 1016—Urée 9.10%; chlorures 9.8%; pas d'albumine, pas d'éléments figurés. Liquide céphalo-rachidien: examen négatif.

Antécédents: 3 avortements, 6 enfants de terme, 1 mortus nato dans une grossesse gémellaire—2 fils sont morts.

(c) *Avec insuffisance Mitrale et Hypertension Artérielle.* Obs. 84 (No. 5658).

Pedro H., 65 ans, homme. Diagn. ophtalm.: O. D. G., glaucome chronique. Diagn. clinique (Dr. J. Rodriguez): Insuffisance mitrale, hypertension artérielle, arythmie extrasystolique—Syphilis.

Examen oculaire (Juillet, 1921): Est malade des yeux depuis deux ans. O. D., ptérigion—chambre antérieure légèrement aplatie; pupille immobile en mydriase moyenne; opacité diffuse du cristallin, empêche examen ophtalmoscopique. Vision = 0. Tension + 2. O. G., légère opacité cornéenne quadrant inf.-interne; pupille immobile mydriatique—Profonde excavation papillaire glaucomateuse atrophique. Vision, perception lumineuse. Tension + 2.

Examen clinique: Arythmie extrasystolique—Légère insuffisance mytrale. Pression artérielle: Mx. 21; Mn. 11. Radioscopie: Coeur augmenté de volume. Réaction de Wassermann négative. Après 3 injections intraveineuses de cyanure (en 6 jours) elle devient positive. Examen d'urine: Densité 1022, indices d'albumine et d'urobiline, urée 19.51%; chlorures 11.4%; quelques globules blancs et rouges, quelques cellules épithéliales polygonales et rondes.

(d) *Avec Aortite, Hypertension Artérielle.* Obs. 92 (No. 5724).

Rosario R. 60 ans, femme. Diagn. ophtalm.: O. D., anophtalmie opératoire; O. G., glaucome inflammatoire chronique. Diagn. clinique (Dr. Borquez): Syphilis—Hypertension artérielle, aortite—Néphrite chronique.

Examen oculaire (Juillet, 1921): O. G., injection péricornéale; cornée de surface chagrinée; chambre antérieure aplatie; pupille mydriatique immobile; fond pas examinable. Vision, perception lumineuse. Tension + 2.

Examen clinique: Battements artériels suprasternaux et supraclaviculaires—choc de la pointe du cœur violent au 5^{ème} espace intercostal (érithisme cardiaque). Pression artérielle (Potain): Mx. 26; au autre jour (Pachon): Mx. 22; Mn. 12. Radioscopie: Crosse aortique dilatée. Réaction de Wassermann négative; réactivation positive. Urine: Densité 1016; albumine 0.10. Urée 9.10; chlorures 8.8; quelques globules rouges, cellules épithéliales polygonales et rondes. Sang: Urée 0.25%.

Antécédents: Souffre fréquemment de céphalées, douleurs rhumatoïdes, troubles gastriques—pas de grossesses.

Obs. 98 (No. 5850)

Louisa S., 74 ans, femme. Diagn. ophtalm.: O. D. G., glaucome absolu. Diagn. clinique: Aortite—Hypertension artérielle—Néphrite chronique—Syphilis.

Examen oculaire: O. D., pupille mydriatique immobile, cristallin opacifié. Vision = 0. Tension + 1; Schiötz 3/7.5 = 36 mm. O. G., pupille mydriatique immobile; excavation papillaire glaucomateuse atrophique, halo péri-papillaire. Vision = 0. Tension + 1; Schiötz 4/7.5 = 31 mm.

Examen clinique: Aorte haute et élargie; renforcement du 2^{ème} bruit aortique—Pouls 108. Pression artérielle (Pachon): Mx. 27; Mn. 16. Radioscopie: Aorte ascendante et descendante augmentée uniformément. La crosse dépasse la clavicule. Grande hypertrophie du ventricule gauche. Réaction de Wassermann positive. Urine: Densité 1017; indices d'albumine. Urée 11.53%; chlorures 9.0%; assez de globules blancs, quelques globules rouges et cellules épithéliales polygonales. Sang: Urée 0.60%. Sphygmogramme: tendance au plateau.

Antécédents: Personnels: sans importance.

Obs. 100 (No. 5896)

Maria Fl., 50 ans, femme. Diagn. ophtalm.: O. D., atrophie bulbaire; O. G., glaucome absolu. Diagn. clinique (Dr. Borquez): Aortite, Néphrite chronique, Hypertension artérielle—Syphilis.

Examen oculaire: O. D., bulbe oculaire atrophique. O. G., vaisseaux ciliaires antérieurs dilatés; chambre antérieure aplatie; pupille en mydriase moyenne, immobile; fond pas examinable. Vision = 0. Tension + 3; Schiötz 0/7.5 = 60 mm.

Examen clinique: Bruit aortique exagéré et systole un peu obscur—Battements suprasternaux et supraclaviculaires. On sent l'aorte derrière la fourchette sternale. Pression artérielle (Pachon): Mx. 22; Mn. 11; pouls 78; respiration 18 par minute. Radioscopie: Aorte très haute et obscure,

légèrement plus grosse—Hypertrophie du ventricule gauche—Sclérose péribronchiale. Réaction de Wassermann négative; réactivation (après 3 injections de mercure) positive. Urine: Densité 1014; albumine 0.20. Urée 8.96%; chlorure 7.0%.

Antécédents: Mari mort l'année passée à l'âge de 40 ans avec une hémiplégie gauche. 1 avortement, 3 enfants vivants.

(e) *Avec Aortite, Hypertension Artérielle et Diabète.* Obs. 96 (de la clientèle privée).

Calixto P., 72 ans, homme. Diagn. ophtalm.: O. D., aphaquie opératoire, glaucome absolu. Diagn. clinique: Diabète—Hypertension artérielle—Aortite chronique—Syphilis.

Examen oculaire (Déc., 1920): O. D., colobome de l'iris opératoire; chambre antérieure profonde; aphakie; excavation papillaire glaucomateuse avec halo péripapillaire. Vision = 0. Tension + 2. O. G., opacité diffuse nucléaire initiale du cristallin, papille normale; pupille et réaction pupillaire normales. Vision 5/50. Tension = 1.

(Novembre, 1921): O. D., idem examen antérieur. Vision = 0. Tension + 1; Schiötz 5/7.5 = 26 mm. O. G., opacité du cristallin partielle. Vision 1/50. Tension = 1; Schiötz 7/7.5 = 18 mm.

Examen clinique (Novembre, 1921): On touche l'aorte derrière la fourchette sternale. Pression artérielle: (7 Nov.) Pachon: Mx. 21; Mn. 9; pouls 72. (16 Nov.) Pachon: Mx. 23; Mn. 8. Radioscopie (Dr. Ducci): Aorte augmentée dans ses trois dimensions—gros coeur. Réaction de Wassermann positive. Urine: Glucose 8.54%.

Antécédents: A été opéré de cataracte O. D. en 1914, mais après l'opération, la vue ne s'était guère améliorée. Souffre d'otite moyenne et salpingite catarrhale bilatérale. (Dr. J. Castro.)

(f) *Avec Myocardite.* Obs. 99 (No. 5880).

Elias M., 45 ans, homme. Diagn. ophtalm.: O. G., panophtalmité secondaire à une ancienne iridectomie antiglaucomateuse; O. D., glaucome absolu. Diagn. clinique (Dr. Rodriguez): Myocardite chronique—Syphilis.

Examen oculaire: Il y a quelques années a été opéré des deux yeux pour glaucome. O. D., colobome opératoire de l'iris; excavation papillaire glaucomateuse atrophique. Vision = 0. Tension + 1. O. G., oedème palpébral; injection bulbaire et quémosis conjonctivale; la conjonctive aux environs de XII montre une tumeur jaunâtre; reflet vert jaunâtre de la pupille; Fond pas examinable. Vision = 0. Tension + 2; Schiötz 0/7.5 > 60 mm.

31 Août: O. D., exentération oculaire avec anesthésie locale, injection 2 cc³ de novocaïne 3% retro-oculaire—abcès du vitré.

Examen clinique: Aorte normale; Tachycardie (pouls 120); Arythmie, respiration 17 par minute; température 36°2. Pression artérielle: Mx. 14.5; Mn. 11 (Pachon): Sphygmogramme athéromateux (plateau). Pouls après quelques jours de digitale: tachycardie et arythmie. Réaction de Wassermann négative; réactivation (après 3 injections mercurielles) positive. Urine: indices d'albumine. Sang: Urée 0.35 %.

Antécédents: La femme a eu 4 avortements, 3 enfants morts dans la première enfance, 5 enfants vivants, sains.

(g) *Avec Myocardite et Aortite.* Obs. 95 (No. 5817).

Anastasie T. 59 ans, femme. Diagn. ophtalm.: O. D., glaucome chronique; O. G., glaucome absolu.

Diagn. clinique (Dr. J. Rodriguez): Myocardite chronique—Aortite chronique—Néphrite chronique hydrurique—Emphysème et sclérose pulmonaire—Syphilis.

Examen oculaire (Août, 1921): O. D., cornée de surface chagrinée; chambre antérieure aplatie; pupille mydriatique; immobile; fond pas examinable. Vision, perception lumineuse. Tension + 2; Schiötz 2/7.5 = 60 mm. O. G., injection péricornéale; cornée chagrinée; chambre antérieure effacée; pupille mydriatique immobile; opacité totale du cristallin. Vision = 0. Tension + 2; Schiötz 1/7.5 = 70 mm.

Examen clinique: Aorte légèrement élevée, élargie, pouls fréquent 96 p. avec intermittences. Pression artérielle (Pachon): Mx. 15; Mn. 8.5. 28 Août: 2ème méditation: Mx. 17.5; Mn. 10.5. 6 Sept.: Mx. 15; Mn. 9; pouls 120, respiration 29, tachycardie et arythmie. 15 Sept.: Après 3 jours de digitale et de theobromine. Pression artérielle: Mx. 16; Mn. 9, pouls 100 arythmie. 20 Sept.: Après 1 gramme de digitale: Mx. 12; Mn. 6.5; pouls 112, arythmie. Radioscopie négative; réaction de Wassermann négative; réactivation (avec 3 injections de cyanure de Hg en 6 jours) positive. Urine: quantité 2050 en 24 heures; densité 1012; indices d'albumine; urée 7.08, chlorures 6.6%; quelques rares globules blancs et cellules épithéliales polygonales. Sang: Urée 0.15%. Liquide céphalorachidien normal.

Antécédents: personnels: pas d'avortements; 11 enfants desquels, 8 morts dans la première enfance.

2. GLAUCOMATEUX SYPHILITQUES.—*Avec Réaction de Wassermann Negative.* Anciens syphilitiques traités.

(a) *Aortite, insuffisance Aortique, Hypertension Artérielle.* Obs. 86 (No. 5690).

Carlota N., 55 ans, femme. Diagn. ophtalm.: O. G., glaucome incipient. Diagn. clinique (Dr. Garavagno): Diabète, aortite avec insuffisance aortique—Hypertension artérielle—Néphrite chronique—Ancienne syphilitique.

Examen oculaire (Juillet, 1921): Depuis 3 mois irritation intermittente de O. G. O. D. G., examen négatif. (A l'oeil droit, près de la papille, il y a une tache blanche rosée, très suspecte . . . Rétinite?) Vision, O. D., O. G. 5/10. Tension, Schiötz: O. D. 7/7.5 = 18 mm. O. G. 4/7.5 = 31 mm.

Examen cardiovasculaire: Coeur augmenté de volume, on touche l'aorte derrière la fourchette sternale—souffle diastolique au 2ème espace intercostal droit. Pression artérielle (Pachon): Mx. 26; Mn. 11. Radioscopie: Aorte haute et grosse—Hypertrophie du ventricule gauche. Réaction de Wassermann négative (après des injections de Mercure). Examen d'urine: Densité 1033; albumine 0.20%; glucose 76.44%; acétona 0.09; urée 28.62; chlorures 10.2; indices d'urobiline; au microscope: rien de pathologique.

Antécédents: En différentes occasions a fait quelques séries de piqûres de mercure.

Obs. 87 (No. 5818)

José M. 57 ans, homme. Diagn. ophtalm.: O. G., glaucome dégénératif; O. D.: glaucome incipient. Diagn. clinique (Dr. Mardones): Syphilis—Insuffisance aortique—Hypertension artérielle—Paralysie faciale droite d'origine centrale.

Examen oculaire (Juillet, 1921): O. D., chambre antérieure un peu aplatie; pupille et fond négatifs. Vision, O. D. 5/5. Tension, Schiötz 5/7.5 = 26 mm. Depuis trois semaines seulement se plaint de douleurs intermittentes à l'O. D. avec irradiations aux dents de ce côté. O. G., cornée en partie opacifiée; chambre antérieure aplatie, pupille en myose pilocarpinique; fond pas examinable. Vision = 0. Tension, Schiötz 0/7.5 = 62 mm.

Examen clinique: Coeur augmenté de volume, bruits un peu étouffés—On touche l'aorte derrière le sternum; souffle diastolique aortique—pouls 76. Pression artérielle (Pachon): Mx. 23; Mn. 12. Radioscopie: Coeur grand et globuleux; aorte assez grosse et obscure, elle touche la ligne articulaire sterno—claviculaire—Clavicules immobiles—Hiles pulmonaires infiltrés. Réaction de Wassermann négative (a reçu des injections mercurielles). Examen d'urine: Densité 1020; albumine 1%; chlorures 13%; pas d'urobiline; au microscope: rien de particulier.

Antécédents: A l'âge de 22 ans, chancre unique avec ganglions non supurés à l'aîne, un mois après laryngite. Quelques mois plus tard, gonorrhée compliquée de cystite, prostatite et orchite. Est arrivé dans l'année dans un service de médecine interne avec dyspnée d'effort, oedème des extrémités, douleurs retrosternales très faibles, palpitations cardiaques. S'est alors amélioré avec régime diétatique et traitement iodo mercuriel. Se plaint de fréquentes amnésies et de vertiges.

3. GLAUCOMATEUX SUSPECTS DE SYPHILIS.—(a) *Hypertension Artérielle*. Obs. 78 (No. 5449).

Françoise E., 60 ans, femme. Diagn. ophtalm.: O. D., glaucome absolu;

O. G., glaucome incipient. Diagn. clinique (Dr. Borquez): Hypertension artérielle, néphrite chronique.

Examen ophtalm. (Juin, 1921): Vision, O. D. = 0; O. G. 5/10. Tension, O. D. > 62 mm.; O. G., 23 mm. (Schiötz); O. D., cornée de surface chagrinée; chambre antérieure aplatie; pupille déformée, mydriatique, immobile; fond pas examinable. O. G., chambre antérieure aplatie; pupille et fond normaux. 19 Juillet: Tension, Schiötz O. G. 5/7.5 = 26 mm.; pupille en mydriase moyenne, avec bonnes réactions; fond négatif.

Examen clinique (Drs. Espildora et Borquez): 2ème bruit aortique renforcé; sous clavière droite élevée; légers battements suprasternaux. Pression artérielle (Pachon): Mx. 22; Mn. 15. Radioscopie: Aorte oblique obscure; hypertrophie du ventricule gauche; pointe rejetée en dehors. Condensation péribronchiale; sclérose pulmonaire peu étendue. Réaction de Wassermann évasive; après réactivation négative. Urine: Quantité 1700 cc³ en 24 heures; densité 1017; indices d'albumine, peu d'indican, traces d'urobiline. Urée 10.40%; chlorures 9.8%; au microscope: globules blancs et cellules épithéliales rares. Sang: Urée 0.48%.

Antécédents: 3 avortements, 7 fils morts dans les premières années de la vie.

(b) *Aortite, Rétrécissement Aortique.* Obs. 79 (No. 5592).

Isabelle C., 60 ans, femme. Diagn. ophtalm.: O. D. G., glaucome chronique. Diagn. clinique: Syphilis, aortite.

Examen ophtalm.: O. D., cornée de surface chagrinée; pupille mydriatique, immobile, déformée; chambre antérieure aplatie; papille vue à travers un nuage, est blanchâtre, excavée. Vision 4/50. Tension, Schiötz 1/7.5 = 51 mm. O. G., pupille déformée, en mydriase moyenne avec réaction paresseuse à la lumière, papille rosée, excavée. Vision 5/30. Tension, Schiötz 5/7.5 = 26 mm.

3 Août: Scléro-iridectomie de Lagrange O. D.

13 Août: Tension, O. D. 2/7.5 = 44 mm.; O. G. 4/7.5 = 31 mm.

27 Août: Tension, O. D. 1/7.5 = 51 mm.; O. G. 5/7.5 = 26 mm.

Examen clinique: Souffle systolique doux à l'aorte. Pression artérielle (Juillet) Pachon, Mx. 14; Mn. 9.5; pression artérielle (1 Août) Pachon, Mx. 18; Mn. 10.5; pression artérielle (27 Août) Pachon, Mx. 18; Mn. 10.5. Radioscopie: Aorte haute, coeur augmenté de volume (pointe rejetée de sa situation normale en dehors). Léger degré de sclérose pulmonaire. Réaction de Wassermann: négative; réactivation de la réaction: négative. Urine: Densité 1026; albumine 0.20; beaucoup d'indican traces d'urobiline; urée 26.02%; chlorures 10.6%; au microscope: globules de pus séparés et en plaques nombreux; peu de globules rouges et de cellules épithéliales rénales; rares cellules vésicales; pas de cylindres. Sang: Urée 0.22%.

Antécédents: Positifs de syphilis. Veuve, n'a pas eu de famille, n'a jamais

été enceinte. Le mari est mort à 35 ans, d'une affection, paraît-il, hépatique; avait la syphilis et l'avait communiquée à sa femme. Celle-ci, au commencement de son mariage, a souffert de maladies vénériennes.

(c) *Avec Aortite, Néphrite Chronique.* Obs. 81 (No. 5630)

Thérèse H., femme, 49 ans. Diagn. ophtalm.: O. D., glaucome initial; O. G., glaucome chronique. Diagn. clinique (Dr. J. Rodriguez): Aortite, néphrite chronique, syphilis.

Examen oculaire (Juillet, 1921): O. D., chambre antérieure un peu aplatie; pupille en myose pilocarpinique; papille normale. Vision 5/20. Tension, Schiötz 5/7.5 = 26 mm. O. G., chambre antérieure aplatie; pupille en mydriase moyenne sans réactions, déformée; papille atrophique avec profonde excavation glaucomateuse. Vision, compte les doigts à 20 cm. Tension, Schiötz 1/7.5 = 52 mm.

Examen clinique: Aorte haute, on la palpe, battements suprasternaux. Pression artérielle (Pachon): Mx. 17; Mn. 8. Radioscopie: Aorte d'épaisseur normale, crosse élevée. Hiles pulmonaires fibreux. Réaction de Wassermann; négative. Urine: Densité; albumine 1.10%; urée 18.21; chlorures 11.6; au microscope: de nombreux globules rouges, peu de globules blancs, peu de cellules épithéliales polygonales; pas de cylindres. Sang: Urée 0.57%.

Antécédents: Syphilitiques positifs. Mari syphilitique—mort à 50 ans.

(d) *Avec Aortite, Hypertension Artérielle, Diabète.* Obs. 91 (cliente particulière).

Juan F., 52 ans, homme. Diagn. ophtalm.: O. D., glaucome chronique ancien; O. G., glaucome chronique initial. Diagn. clinique (Dr. J. Rodriguez): Ancien diabétique, grande hypertension artérielle, aortite, syphilis ou tuberculose pulmonaire?

Examen oculaire: A été opéré, il y a peu de mois de O. D.; depuis quelques semaines gêne à O. G. O. D., pupille déformée, pyriforme, sans iridectomie périphérique visible; petit kyste conjonctival à XII en face de sclérectomie; papille blanchâtre avec excavation profonde centrale. Vision 5/10. Tension = 1, Schiötz 9/7.5 = 14 mm. O. G., examen négatif. Vision 5/5. Tension + 1, Schiötz 3/7.5 = 36 mm.

Examen clinique: Hypertrophie du cœur—Dilatation de l'aorte (aortite chronique), renforcement du 2ème bruit aortique; bruit de timbre métallique; infiltration portion moyenne du poumon gauche; tuberculose ou syphilis pulmonaire? Pression artérielle (Pachon): Mx. > 35 mm.; Mn. 9; pouls 110. Radioscopie: Aorte grosse et haute—Hypertrophie du cœur. Urine: Densité 1019; quantité 2800 gr. en 24 heures. Indices d'albumine, indices de mucine, glucose 25.10; urée 17.40; acide urique 0.42; phosphates 1.06; chlorures 5.60; un peu d'excès de leucocytes et quel ques cellules rondes. Réaction de Wassermann plusieurs fois négative en différentes occasions.

Antécédents: 1 avortement, 4 fils sains. Il y a un an forte attaque de grippe, depuis lors, il dit avoir le poumon faible. L'étiologie de l'affection pulmonaire est encore en étude.

IV. GLAUCOMATEUX ARTERIOSCLEREUX

(32 Cas)

Nous avons estimé que l'affection vasculaire de nos malades de près de 60 ans pouvait être considérée comme obéissant au procès de sclérose sénile, s'il n'existait pas une autre étiologie capable d'expliquer l'altération des vaisseaux.

Le Glaucomateux artérioscléreux, non syphilitique, a été observé 32 fois sur 100 malades.

Des 100 glaucomateux étudiés, 41 avaient plus de 60 ans et de ceux-ci 24 étaient artérioscléreux et 13 syphilitiques.

Tout en donnant une valeur relative à notre petite statistique, on pourrait dire que:

Si avant 50 ans le glaucomateux est presque toujours syphilitique, après les 60, on trouve chez lui plus souvent l'artériosclérose que la syphilis.

Comme chez les syphilitiques, l'hypertension artérielle et la lésion aortique est d'une grande fréquence: Hypertension—25 fois sur 32 cas; lésion de l'aorte 17 fois.

Le rétrécissement aortique a été trouvé 2 fois.

Le compromis du rein n'est pas rare, mais nous répétons ici la même advertance que précédemment, l'examen rénal a été par trop superficiel dans presque toutes nos 65 premières observations.

Une fois, la néphrite était accompagnée de diabète (obs. 15).

GLAUCOMATEUX ARTERIOSCLEREUX.¹—(32 cas.) (Les observations contenues dans ce Mémoire apparaissent soulignées.)

- (a) Avec signes vasculaires périphériques légers; obs. 2, 17.
- (b) Avec symptômes cardio-vasculaires frustes; obs. 68, 72, 74, 94.
- (c) Avec hypertension artérielle; obs. 1, 24, 64, 60, 77, 97.
- (d) Avec hypertension artérielle et néphrite chronique; obs. 67.
- (e) Avec hypertension artérielle et image radioscopique anormale; obs. 10.
- (f) Avec hypertension artérielle, image radioscopique anormale et légère néphrite chronique; obs. 68.

¹ Les observations de 1 à 75 forment partie du Mémoire présenté à la Société d'Ophthalmologie de Paris.

(g) Avec hypertension artérielle et athérome aortique; obs. 5, 7, 27, 29, 33, 35, 38, 43, 70, 85, 88.

(h) Avec hypertension artérielle et mitro-aortisme; obs. 83.

(l) Avec athérome et rétrécissement aortique; obs. 59.

(m) Avec athérome aortique, hypertension artérielle et néphrite chronique; obs. 15, 58, 73.

(n) Avec athérome et rétrécissement aortique, hypertension artérielle, néphrite chronique; obs. 61.

II. OBSERVATIONS DE GLAUCOMATEUX ARTERIOSCLEREUX

Contenues dans ce mémoire

(a) Avec hypertension artérielle; obs. 60, 77, 97.

(b) Avec hypertension artérielle et mitro-aortisme; obs. 83.

(c) Avec hypertension artérielle, athérome aortique, arythmie; obs. 85, 88.

(d) Avec symptômes vasculaires frustes; obs. 94.

(a) *Avec Hypertension Artérielle.*—Obs. 60 (No. 5849).

Cas considéré comme négatif dans notre communication à la Société d'Ophthalmologie de Paris. La malade étant revenue se présenter à notre clinique au mois d'Août 1921, nous avons pu constater alors un franc compromis du système vasculaire général.

Aurora D., 63 ans. Diagn. ophtalm.: O. D. G., glaucome chronique. Diagn. clinique: Cas négatif, Sept. 1920: hypertension artérielle, néphrite chronique hypertensive. (Août, 1921.)

Examen oculaire (Sept., 1920): O. D., pupille mydriatique avec bonnes réactions, milieux transparents normaux; papille avec excavation glaucomateuse rose clair, halo péripapillaire. Vision 5/7.5. Tension + 1. O. G. Chambre antérieure trouble; pupille mydriatique immobile; papille atrophique avec excavation glaucomateuse; halo péripapillaire. Vision, perception lumineuse. Tension + 2.

Examen clinique (Sept., 1920): Aorte et coeur normaux. Radioscopie négative. Réaction de Wassermann négative. Urine: Ex. négatif? (douteux).

Examen oculaire (Août, 1921): Diagn. ophtalm.: O. D. G., glaucome absolu. O. D. G., profonde excavation glaucomateuse atrophique, entourée d'un halo péripapillaire; pupilles immobiles, déformées en mydriase moyenne. Vision, O. D. G. = 0. Tension, Schiötz: O. D. = 71 mm.; O. G. = 60 mm.

Examen clinique (Août, 1921), (Dr. Borquez): 2ème bruit aortique renforcé. Pression artérielle (Potain): 22 au lieu de 15 à 18; 2ème examen (Pachon): Mx. 20; Mn. 11 (Dr. Prado); athérome aortique. Urine: Densité

1017; indices d'urobiline; albumine 0.20; chlorures 7.8; Urée 16.35%; globules de pus séparés et en plaques nombreux; quelques globules rouges; quelques cellules épithéliales polygonales et rondes.

Obs. 77 (No. 5498)

Marguerite C., 65 ans, femme. Diagn. ophtalm.: O. D. G., glaucome chronique. Diagn. clinique (Dr. Gonzalez Cortés): Hypertension artérielle.

Examen ophtalm. (Juin, 1921): Diminution de la vue survenue brusquement il y a plus d'un mois accompagnée de grandes douleurs frontales. O. D. G., injection périkératique, anesthésie cornéenne; pupilles mydriatiques, déformées, immobiles; fond pas examinable (à l'O. D. on distingue sans détails la papille). Vision, O. D. = 0; O. G., mouvements de la main à 0 m. 50. Tension, O. D., O. G. + 2.

24 Juin: Opération O. D. sclero-iridectomie sans incidents.

13 Septembre: Tension, Schiötz O. D. 8/10 23 mm.; O. G. 5/10 37 mm.

Examen clinique: Cœur et aorte négatifs. Pression artérielle (Pachon): Mx. 20; Mn. 13. Sphygmogramme: Tendance au plateau. Radioscopie: négative. Réaction de Wassermann négative. Urine: Quantité 2000 gr. en 24 heures. Densité 1014; indices d'albumine, peu d'indican, traces d'urobiline; urée 6.50%; chlorures 10.6%; au microscope; quelques globules rouges; des globules blancs et des cellules épithéliales polygonales rares; pas de cylindres. Sang: Urée 0.45%. 28 Septembre: Pression artérielle (Pachon): Mx. 18; Mn. 10.5.

Antécédents: Familiaux et personnels sans importance.

Obs. 97 (No. 5772)

Gracia F., 70 ans, femme. Diagn. ophtalm.: O. D., glaucome inflammatoire chronique; O. G., glaucome absolu. Diagn. clinique (Dr. Rodriguez): Athérome aortique—Hypertension artérielle.

Examen clinique: On touche l'aorte derrière la fourchette sternale. Pression artérielle (Août) Pachon, Mx. 17; Mn. 12; pression artérielle (13 Sept.) Pachon, Mx. 19; Mn. 12.5; pression artérielle (28 Sept.) Pachon, Mx. 16; Mn. 9.5. Radioscopie: Aorte haute, obscure, de grosseur normale. Condensation péribronchiale. Sphygmogramme athéromateux (plateau). Réaction de Wassermann négative; réactivation négative. Urine: Densité 1015; indices d'albumine; urée 13.01; chlorures 6.2%; quelques globules blancs et cellules épithéliales polygonales. Sang: Urée 0.25%.

Antécédents personnels Sans importance.

Examen oculaire: O. D., injection bulbaire, chambre antérieure aplatie; pupille en mydriase moyenne, déformée, sans réactions, fond pas examinable. Vision, perception lumineuse. Tension + 2, Schiötz > 60 mm. O. G., opacité cornéenne légère, pupille mydriatique immobile; chambre antérieure aplatie; fond pas examinable. Vision = 0. Tension + 3 Schiötz > 60 mm.

(b) *Avec Hypertension Artérielle et Mitro-aortisme.*—Obs. 83 (No. 5678).

Rosario A., 64 ans, femme. Diagn. ophtalm.: O. D., glaucome inflammatoire absolu. Diagn. clinique (Dr. Borquez): Mitro-aortisme, hypertension artérielle.

Examen oculaire (Juillet, 1921): Depuis un mois douleurs oculaires droites violentes et névralgies faciales. O. D., injection bulbaire intense; pupille mydriatique sans réactions; iris décoloré; cornée chagrinée avec sensibilité très diminuée; excavation papillaire glaucomateuse. Vision = 0. Tension + 2, Schiötz 1/7.5 = 57 mm. O. G. chambre antérieure aplatie; papille avec excavation physiologique. Vision 5/30. Tension = 1, Schiötz 9/7.5 = 14 mm.

Examen clinique: Battements suprasternaux et sous-clavières visibles; coeur augmenté de volume, pointe en dehors de la ligne mamellaire—Souffle systolique mitral et souffle marqué, un peu rude systolique au foyer aortique. Pression artérielle (Pachon): Mx. 21.5; Mn. 9; un autre jour (Potain): Mx. 20; pouls 88. Radioscopie: Adhérences costodiaphragmatiques droites—Examen aorte et coeur négatif. Examen d'urine: indices d'albumine. Sang: Urée 0.30%. Réaction de Wassermann négative.

Antécédents syphilitiques: Négatifs

(c) *Avec Hypertension Artérielle, Athérome Aortique, Arythmie.* Obs. 85 (No. 5451).

Manuel G., 80 ans, homme. Diagn. ophtalm.: O. D. G., glaucome chronique; dacryocystite gauche. Diagn. clinique (Dr. Rodriguez): Athérome aortique, Arythmie, Hypertension artérielle.

Examen oculaire (Mai, 1921): O. D. G., en mydriase moyenne avec réactions pupillaires paresseuses à la lumière, bonne à la convergence; papilles blanches atrophiques avec excavation pas profonde; champ visuel un peu rétréci. Vision, O. D. 5/15; O. G. 5/40. Tension, Schiötz O. D. 2/7.5 = 43 mm.; O. G. 4/7.5 = 31 mm.

Examen clinique: Thorax emphysémateux; aorte haute, on la touche derrière la fourchette sternale; 2ème bruit aortique renforcé; coeur augmenté de volume; arythmie; le poumon droit ne respire pas bien à la base.—On touche le bord inférieur du foie. Pression artérielle: Mx. 23; Mn. 12.5. Radioscopie: Aorte haute et obscure, coeur légèrement augmenté de volume, sclérose pulmonaire. Réaction de Wassermann: négative. Examen d'urine: Densité 1021; indices d'albumine; urée 20.19%; chlorures 12.6%; au microscope: peu de globules rouges et blancs. Sang: Urée 0.15%.

Obs. 88 (No. 5711)

Marie T., 56 ans, femme. Diagn. ophtalm.: O. D., glaucome incipient; O. G., glaucome absolu. Diagn. clinique (Dr. Perez Canto): Hypertension artérielle; aortite, néphrite chronique.

Examen oculaire (Août, 1921): Souffre de l'oeil gauche depuis 4 ou 5 mois et dernièrement voit moins bien avec l' O. D., perception de cercles colorés. O. D., chambre antérieure aplatie; pupille en mydriase moyenne, avec réaction pupillaire lumineuse paresseuse, réaction de convergence normale. Examen ophtalm.: négatif. Vision, O. D. 5/10. Tension, Schiötz 5/7.5 = 26 mm. O. G., cornée chagrinée avec opacités; chambre antérieure pas aplatie, pupille mydriatique immobile; fond pas examinable. Vision = 0. Tension, Schiötz > 1/7.5 > 52 mm.

Examen clinique: Tachycardie (100 p.) aorte légèrement dilatée transversalement; palpation douloureuse des carotides. Pression artérielle (Pachon): Mx. 23; Mn. 14, à la radiale gauche; pression artérielle (Pachon): Mx. 21; Mn. 14, à droite. Radioscopie: Aorte haute et grosse; hypertrophie du ventricule gauche. Réaction de Wassermann négative; réactivation négative. Examen d'urine: Densité 1024; albumine 0.20%; urée 15.27%; chlorures 12.4%; traces d'urobiline; au microscope; globules de pus séparés et en plaques rares; globules rouges très rares; peu de cellules épithéliales polygonales et rondes.

Antécédents: 2 fils sains, pas d'avortements ni de mortus nato.

(d) *Avec Symptômes Vasculaires Frustes.* Obs. 94 (No. 5707).

Toribio P., 68 ans, homme. Diagn. ophtalm.: O. D. G., glaucome absolu. Diagn. clinique: Artériosclérose.

Examen oculaire: O. D. G., chambres antérieures un peu effacées; pupilles déformées, mydriatiques, immobiles; excavation glaucomateuse atrophique. Vision, O. D. G. = 0. Tension, Schiötz, O. D. G. 1/7.5 = 51 mm.

Examen clinique: Bruits valvulaires de ton métallique. Pression artérielle (Pachon): Mx. 14.5; Mn. 7. Radioscopie: Aorte dilatée uniformément—Hypertrophie du ventricule gauche. Sphygmogramme athéromateux. Réaction de Wassermann négative. Urine: Densité 1014; indices d'albumine; urée 8.96; chlorures 7.8; rares globules blancs et cellules épithéliales polygonales.

V. GLAUCOMATEUX AVEC SYMPTOMES CARDIOVASCULAIRES D'ÉTILOGIES DIVERSES

Rhumatisme—Obs. 75

Obésité —Obs. 69

Douteuse —Obs. 36, 89

Obs. 89 (No. 5696)

Augustin R., 45 ans, homme. Diagn. ophtalm.: O. D., glaucome absolu. Diagn. clinique: Néphrite chronique incipiente? (Dr. Garcés.)

Examen oculaire: O. D., pupille déformée, sans réaction lumineuse, papille atrophique avec excavation glaucomateuse. Vision = 0. Tension, Schiötz 2/7.5 = 43 mm. O. G., négatif. Vision 5/10. Tension 7/7.5 = 19 mm.

Examen clinique: Coeur et aorte négatifs, cependant bruits du coeur étouffés, systole âpre. Pression artérielle (Pachon): Mx. 13; Mn. 7. Sphygmogramme athéromateux (plateau). Radioscopie négative. Examen d'urine négatif. Sang: Urée 0.35%. Réaction de Wassermann négative; réactivation négative. Constante d'Ambard positive (Dr. Garcés). Urée dans le sang (ur) 0.59. Urine produite en 60 minutes 61 c.c.; urine en 24 heures (selon calcul) 1464. Urée dans l'urine ‰; (C) 17.93; urée dans l'urine en 24 heur; (D) 26,²⁵. Poids du malade; (P) 66.

$$K = \sqrt{\frac{0.59}{26.25 \times \frac{70}{66}}} \times \sqrt{\frac{17.93}{25}} = 1.09$$

(Normal 0.042 à 0.074.)

VI. GLAUCOMATEUX A EXAMEN CARDIOVASCULAIRE NEGATIF

Antécédent clinique:	<i>grippe</i>	—Obs. 44
“	“ <i>sénilité</i>	—Obs. 47, 65, 76, 80
“	“ <i>aucun</i>	—Obs. 49
“	“ <i>de syphilis</i>	—Obs. 90

Obs. 76 (No. 5478)

Manuel C., 61 ans, homme. Diagn. ophtalm.: O. D., glaucome chronique inflammatoire, cataracte; O. G., glaucome absolu. Diagn. clinique: Artériosclérose sénile incipiente?

Examen ophtalm. (28 Mai, 1921): Il y a huit ans qu'il ne voit pas avec O. G. Depuis quatre ans sa vue de O. D. a baissé progressivement. O. D., injection périkeratique; chambre antérieure effacée; pupille déformée, mydriatique, immobile; opacité du cristallin. Vision, perception lumineuse. Tension, Schiötz 1/10, 70 mm. O. G., injection périkeratique légère; chambre antérieure aplatie; pupille mydriatique immobile; excavation glaucomateuse atrophique profonde. Vision = 0. Tension, Schiötz 3/10, 52 mm.

Examen clinique: Pouls 76, radiale situation anormale, inspection et palpation négative, renforcement du 2ème bruit aortique. Coeur examen négatif. Pression artérielle (Pachon): Mx. 18; Mn. 9. Sphygmogramme (humeral) tracé suspect d'hypertension. Radioscopie: Sommets pulmonaires obscurs; infiltration fibreuse des poumons; aorte et coeur normaux. Réaction de Wassermann négative. Examen d'urine: Densité 1017; indices d'albumine; un peu d'indican; traces d'urobiline; urée 7.80‰; chlorures 8.6‰; au microscope: globules blancs et cellules épithéliales polygonales rares.

Obs. 80 (No. 5587)

Romualdo A., 62 ans, homme. Diagn. ophtalm.: O. D. G., glaucome chronique. Diagn. clinique: Léger degré d'emphysème pulmonaire—Pas de lésions vasculaires perceptibles.

Examen ophtalm.: O. D. G., chambre antérieure aplatie; iris normal; pupille sans réaction lumineuse, légèrement mydriatique; papille blanchâtre, excavée. Vision, O. D., perception lumineuse; O. G., compte les doigts. Tension, Schiötz O. D. $2/7.5 = 43$ mm.; O. G. $3.5/7.5 = 33$ mm.

Examen clinique: Aorte haute et élargie? coeur normal. Pression artérielle (Pachon) Mx. 17.5, Mn. 7.5 (on n'a pas fait de nouvelles méditations). Réaction de Wassermann négative; (on n'a pas fait de réactivation). Radioscopie: Aorte et coeur normaux. Sclérose pulmonaire, léger degré d'emphysème. Urine: Densité 1025; indices d'albumine; beaucoup d'indican, traces d'urobiline. Urée 16.91%; chlorures 15.2%; au microscope globules blancs et rouges rares; peu de cellules épithéliales polygonales; pas de cylindres; de rares cristaux de phosphate amonio-magnésien.

Obs. 90 (No. 4507)

Macario A., homme, 62 ans. Diagn. ophtalm.: O. D. G., glaucome chronique, avec atrophie papillaire. Diagn. clinique: Cas négatif, syphilis douteuse?

Examen oculaire (Novembre, 1919): O. D. G., pupilles en mydriase moyenne avec bonnes réactions; profonde excavation glaucomateuse atrophique; champ visuel rétréci en dedans. O. D. > O. G. Vision, O. D. 5/30; O. G. 5/10. Tension, O. D. = 1; O. G. + 2. 2ème Examen (Juillet, 1921). O. D. G., pupilles mydriatiques avec réactions pupillaires très paresseuses; atrophie papillaire avec excavation glaucomateuse profonde. Vision, O. D. main à 0.m.30; O. G. 5/20. Tension, Schiötz: O. D. 31 mm.; O. G., 37 mm.

Examen clinique: Cardiovasculaire négatif. Pression artérielle (Pachon): Mx. 18; Mn. 7. Radioscopie négative. Sphygmogramme: Normal. Réaction de Wassermann négative; réactivation négative. Examen d'urine: Négatif—(urée 24.34, chlorures 15.2%). Constante d'Ambard: Normale 0.056 (Dr. Garcés). Réaction de Wassermann chez la femme négative.

Antécédents cliniques de syphilis positifs. Marié 2 fois—Du premier lit 8 enfants, 6 vivants, 1 avortement, 1 mortus-nato—Du 2ème lit, le premier et le 3ème enfants mortus nato, 5 vivants.

VII. CONSIDERATIONS PATHOGENIQUES—CONCLUSIONS CLINIQUES

Nous venons de constater que sur 100 malades de glaucome primitif examinés au point de vue général, 90 présentaient des symptômes vasculaires.

On accepte d'autre part aujourd'hui que dans le glaucome la lésion anatomo-pathologique primordiale, et peut-être initiale, serait la lésion du vaisseau oculaire.

Si on relationne ces deux faits, on voit aussitôt qu'il doit exister une parenté très proche entre eux.

La clinique en nous dévoilant l'hypertension artérielle, l'aortite, la

myocardite, etc., . . . sur le glaucomateux, nous oblige d'abord à accepter au moins une parenté anatomique entre l'affection oculaire et l'affection générale, puisque dans les deux affections c'est toujours l'arbre vasculaire qui est malade, que ce soit dans le tronc ou dans ses branches.

La Biologie, de son côté, nous dit qu'une lésion vasculaire déterminée est un procès toujours le même, absolument identique, soit qu'il se déroule sur le vaso-vasorum de l'aorte, de l'artère radiale ou sur les capillaires de l'oeil. Il existerait donc aussi entre le procès vasculaire oculaire et général une parenté pathologique.

L'hypothèse qui fait dépendre, en premier lieu, l'hypertension oculaire de la lésion du vaisseau, n'est qu'une hypothèse, mais la fréquence extrême du compromis du système vasculaire général chez le glaucomateux, donne un puissant appui à cette théorie pathogénique.

Si cette théorie était dans le vrai, l'étiologie du glaucome, problème si obscur, s'éclairerait d'une façon inattendue.

En effet, il serait logique alors d'admettre qu'une seule cause a dû toucher la paroi vasculaire, ici et là, le vaso-vasorum de l'aorte et le réseau ophtalmique, malades simultanément.

L'examen clinique, l'étude des antécédents, la Réaction de Wassermann, etc., . . . donnent l'étiologie de l'hypertension artérielle, de la myocardite, de l'aortite—on arriverait dans le glaucome, par un chemin identique, à la solution du problème.

En d'autres termes, le glaucome aurait la même signature étiologique que la cardiopathie qui l'accompagne.

Mais refusons toute valeur à ces considérations pathogéniques, comme étant de simples vues de l'esprit non démontrées.

“Le Glaucomateux n'est pas seulement un malade des des yeux, puisque dans un 90% des cas il souffre en même temps d'une autre affection.”

Le médecin a le devoir de chercher à dépister cette autre maladie par une investigation clinique minutieuse; l'examen oculaire doit donc se compléter par l'examen général.

Et ceci fait, le traitement local hypotensif doit aussi se compléter par la thérapeutique générale—propre à chaque cas.—Santiago, 1^{er} Decembre, 1921.

THE DIAGNOSIS OF GLAUCOMA

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London, England

To present a paper on the diagnosis of glaucoma before an International Congress is a task entirely different from that of writing on the same subject for the purposes of a text-book, since a mass of detail required by the student is better omitted, attention being directed to the less well-known or more debatable departments of the subject. Another point to be borne in mind is that diagnosis helps us in two somewhat different directions: (1) To ascertain whether glaucoma is present or not and (2) to enable us to decide whether to adopt therapeutic or operative measures in the treatment of individual cases.

There are few problems confronting the ophthalmic surgeon which are more difficult than that of the diagnosis of a pathologic rise of tension in certain cases; and yet the writer would desire to anticipate what is yet to come by the very emphatic statement of his belief that a surgeon, who will take the trouble to avail himself to the full of the modern methods of diagnosis, need never be long in doubt as to the presence or absence of glaucoma. It is true that a correct diagnosis can sometimes only be made by waiting; but if the surgeon is alive to the condition present, and loses no time in collecting the necessary data, it may be questioned whether the delay involved need ever lead to serious consequences. The same remarks are true of the measures and observations necessary to enable us to come to a sure conclusion as to whether a condition of glaucoma is progressive or stationary. Few systems are more evil than that of the collective consultation, in which a number of surgeons meet, conduct a superficial examination, and assure the patient that he should, or should not, be operated upon. Such a method appeals powerfully to the patient and his friends. It is popular, dramatic, and worthless. The diagnosis of a difficult case of glaucoma, whether the point at issue be the presence of increased tension or the question of the progress of the disease, can only be established by the work of an individual surgeon, who conducts a routine and careful examination, to which he is willing to devote unlimited

time. When all his data are available, he may perhaps with profit lay them before one or more colleagues and get the latter to check doubtful points which his work has revealed. This is not the method of the sensational novelist or the equally sensational newspaper paragraph writer, or, only too often, of the friends who prefer the consultation of a number of surgeons; but it is the one which best subserves the interests of the patient.

With this introduction, we shall now take up a number of points in turn.

THE CONJUNCTIVA AND SCLERA

It is well known that circumcorneal congestion, which may be of a very pronounced type, is a constant accompaniment of acute and sub-acute glaucoma, while enlargement of the episcleral vessels, and especially of those which perforate the tunic of the eye, is the rule in chronic cases. It has been suggested that these vessels are arteries. The writer has no hesitation in asserting that they are veins: (1) They have the appearance and color of veins; (2) when cut across in the course of an operation, their bleeding is typically venous; (3) when the lumen of one of these vessels is closed at each of its ends as far apart as possible, it not only fills up from either end indifferently when the pressure is released, but it actually fills from lateral feeder vessels even while the two ends are still blocked; (4) under high magnification with a corneal microscope and Gullstrand slit-lamp, no pulsation whatever can be observed in any of these trunks.

THE CORNEA

The steaminess of this membrane in acute cases and the secondary changes in late cases are too familiar to need more than a mention. There are, however, two symptoms due to the steaminess of the cornea which deserve notice, viz., mists and halos. The great importance of these lies, not merely in the fact that they attract the patient's attention very markedly, but still more in that they serve as a very delicate test of the recurrence and duration of exacerbations of the disease. Those who are suffering from simple glaucoma, even with mild congestive intervals, may never be conscious either of mists or of halos, but the victims of acute and subacute attacks need never be in any doubt as to the recurrence of their trouble, once they have been trained to employ this test.

It is important to distinguish two quite different sets of conditions under which mists and halos are observed, viz., (1) in the early morn-

ing, and (2) at those times of the day or night when fatigue is most pronounced. The early morning symptoms are due to the failure during the sleeping hours of the pump action, described by Professor Thomson, and exerted on Schlemm's canal through the action of the ciliary and iris muscles. Those which come on late in the day, as the result of fatigue, are to be attributed to vascular congestion and are frequently relieved by food or rest. They, in fact, constitute the incursion into the case of the vascular factor, as an outcome of disturbances of the vasomotor equilibrium.

MISTS OF VISION.—It is important to remember that, though these are undoubtedly due in part to edema of the corneal epithelium and of the superficial layers of the cornea (and possibly also of the endothelium lining Descemet's membrane), a similar condition of the retina almost certainly plays an important part in the production of the symptom. Such a condition is predisposed to by the cutting off in some measure of the arterial blood supply, and by the obstruction to venous return, both being factors which interfere with the due nutrition of the retina.

HALOS.—These are best seen when looking at bright lights in the dark. When they are present, the ordinary patient can see them excellently around a match or candle flame held at arm's length. In the dark-room the glow of a self-lit ophthalmoscope bulb—especially when not too bright—gives very vivid halos.

It is most important that our patients should understand what we mean by halos, since so many conditions may easily be confused with the true colored rings seen by the glaucomatous subject. For this purpose a glass plate dusted with lycopodium should be kept handy; once a patient has looked through this, he knows for all time exactly what we mean when we ask him whether he has seen colored rings.

The following conditions are from time to time mistaken for halos: (1) The golden haze made up of radiating luminous beams which a normal eye may see surrounding a light in the dark, and which is often dissipated by rubbing or drying the eye. (2) Imaginary halos seen by nervous people who have heard glaucoma discussed. (3) The scintillating scotoma of migraine. (4) The rapidly closing in rings of color seen by nervous or overtired people when they shut their eyes in the dark. (5) The halos due to damage to the corneal epithelium produced by caustic and other medicinal applications. (6) The transient halos complained of by those who suffer from conjunctivitis. These are associated with mucous discharge, and they at once disappear on

washing the eye; they are probably due to the inclusion in the sticky mucus of masses of leukocytes imprisoned in the conjunctival exudate. (7) The halos seen when looking at a bright light far back in a big room through a steamy plate-glass window from the dark outside; these are the same as those seen through breathed on or steamy spectacle glasses. (8) The halos seen by some patients in the early stage of cataract; these are said to disappear if looked at through a pin-point diaphragm. (9) The physiologic halos which any normal eye can see if carefully looked for. These are said to be brighter and more prominent in the old. (10) The physiologic halos seen by many normal eyes when the pupils are dilated.

The majority of these conditions can be easily differentiated from the glaucomatous halos by any surgeon who is acquainted with the fact that they may possibly be seen by non-glaucomatous people. A few inquiries will soon show their true nature. There are, however, some points which may here be profitably emphasized. The real glaucoma halo is much brighter than its imitators; the patient can very easily recognize at least three colors, a central blue, a middle yellow, and an outer orange red, or orange. In addition a band of green can often be detected between the blue and yellow. The diameter of the glaucomatous halo varies considerably in different patients, and even in the same patient at different times. I have never found it less than 6.50° or more than 11.54° . The physiologic halos, on the other hand, are stated by authorities to measure constantly about 7° in diameter. It has already been mentioned that the colored rings are very feeble, requiring close attention for their recognition. The fact that a patient sees a colored halo after the use of a mydriatic may easily lead to the suggestion that the surgeon has been guilty of negligence in the use of the drug. The danger is all the greater since many members of the public are better informed on the subject of glaucoma halos than the majority of the medical profession. It is therefore essential that in every such case we should take note of the diameter of the halos, of their brilliance, of the colors recognized, and of the tension of the eye at the time. By doing so we may save a colleague much undeserved discredit.

So important did this subject appear to the writer, that he sought the assistance of Mr. H. H. Emsley, of the Northampton Polytechnic Institute, Clerkenwell. This gentleman has most kindly spent a great deal of time and trouble over the matter. He points out that Druault's test, as suggested by Morax, is not quite accurate. If a true

glaucoma halo, or any other halo, due to interference with the corneal tissues is observed, and at the same time a straight-edged screen (a stenopeic slit held vertically, does excellently) is moved across the area of vision close to the eye, the ring of the halo is obliterated just in proportion as the eye is covered by the slit. If, on the contrary, we observe a physiologic halo—and this is much easier with a dilated pupil—and if we now move our straight-edged screen across the area of the field of vision, an entirely different phenomenon is seen, due to the fact that the halo is here produced by the grid-like criss-crossing of the lens fibers. As the screen comes across the eye, instead of seeing the complete halo, we now only see two rather narrow spectral bands, one at each end of a diameter of the halo; with the movement of the screen, these swing round like the spokes of a cart-wheel, traveling clockwise or counter-clockwise alternately, according as the screen is moved in one or the other direction. This beautiful little experiment of Mr. Emsley's enables us to tell at once whether the halos—whether seen spontaneously by the old, or as a result of mydriasis in younger people—are produced by the lens fibers, or are truly corneal. In the former case they have no connection with the intraocular pressure; in the latter, they may or may not have such a connection.

A last word as to the measurements of halos: With the light 10 feet from the observing eye, the diameters of the halos will be approximately as follows: For 4°, 8.5 inches; for 5°, 10.5 inches; for 6°, 12.5 inches; for 7°, 14.75 inches; for 8°, 17 inches; for 9°, 19 inches; for 10°, 21 inches; for 11°, 23.33 inches; for 12°, 25.35 inches.

THE ANTERIOR CHAMBER

SHALLOWING OF THE ANTERIOR CHAMBER.—This well-known sign may occur under two quite different conditions: (1) It may be the result of an overdistention of the vitreous body, of congestion of the ciliary body, or of both; or (2) it may result from the sealing down of the angle of the chamber, due to the adhesion of the iris base to the corneal periphery.

In either case, and especially in the latter, it may be important to be able to measure the depth of the chamber. Much information may thus, possibly, be obtained as to the progress a case is making on the downward road, and as to the prospect of an operation proving a success. With regard to the latter point, the writer holds strongly that an iridectomy is useless once the angle is sealed down, unless it accidentally gives rise to a filtering scar. If this be accepted, it is

obvious that we should aim at performing a filtering operation to start with, and should not leave our main object to be the sport of chance. Again, if we desire to trephine, it is of importance to know beforehand how far forward the obliteration of the angle has progressed, and, if we have reason to believe that it is very far forward, we should get all the corneal splitting we safely can.

Messrs. Zeiss have fitted to their corneal microscope a device known as "the Ulbrich drum." With the aid of this, and by a simple calculation, the depth of the chamber can easily be measured. Even apart from any calculation, the relative depths of the same chamber can be estimated from time to time; this is probably the most important element in the case.

These considerations have been put forward because it seems probable that important advances can be made if such lines of work are followed up; and America is far more likely to do the work than most parts of Europe, for the present at least.

CLOUDINESS OF THE ANTERIOR CHAMBER, OR THE PRESENCE OF DEPOSITS ON THE ANTERIOR SURFACE OF THE IRIS, OR ON THE POSTERIOR SURFACE OF THE CORNEA.—These are, in reality, signs of an iridocyclitis; their observation is most important, as the diagnosis of the cause of the glaucoma, and the main indication for the exhibition of mydriatic drugs hang on their recognition. This point was well brought out in the valuable discussion before the American Ophthalmological Society, at their Fifty-fifth Annual Meeting in Atlantic City in 1919.

KOEPPE'S SIGN.—Koeppé has claimed that glaucoma can be recognized, even in what he terms "the pre-glaucomatous stage," by the aid of the Gullstrand slit-lamp—and this months or even a year before any other sign of the disease has appeared. He states that as a result of the morbid changes, which are taking place, pigment granules are set free, and that these wander out in the form of a fine dust, and are to be seen on the surface of the iris stroma. The observation has been both supported and contradicted. The writer has certainly seen such granules in glaucoma cases, but, he has also seen unquestioned cases of established glaucoma, in which no trace of them can be detected. The matter is one of great interest, and as some American ophthalmologists have devoted their attention to the subject, it is to be sincerely hoped that some further light will be shed on it at the Congress. If we could, by this or by any other means, anticipate a certain diagnosis of progressive glaucoma, it would be of incalculable

benefit to us, for we could then undertake a well-planned operation at a date when everything is in favor of success, instead of being forced to resort to surgery at a dangerous time, as is now only too often the case.

THE CILIARY BODY

The impairment of accommodation which accompanies glaucoma is too well known to require more than a passing mention. The appearance of a manifest hyperopia is less widely recognized as a sign of the disease, although it is far from being uncommon. Its genesis is easily explained: The original condition in such cases was one of latent hyperopia; under the paralyzing influence of the increase of pressure, acting upon the third nerves and on their terminals in the ciliary muscles, the hyperopia has become manifest.

THE LENS

Much has been written recently on the subject of cataract complicating glaucoma. It is necessary to distinguish sharply between 3 conditions which are too often confused with each other:

(1) The form of *cataract secondary to glaucoma* is characterized by a want of definition in its appearance. It looks like a smoky, greenish or bluish haze, and does not present the definite features with which we are all familiar in an ordinary primary cataract.

(2) *Glaucoma Secondary to Cataract*.—The appearance of the lens in the majority of these cases is characteristic of the intumescent variety, the history is unmistakable, and the presence of a primary and hitherto uncomplicated cataract in the opposite eye clinches the diagnosis.

(3) *Cataract occurring as an accidental complication of glaucoma* is by no means uncommonly met with by any surgeon in large practice.

The so-called "green reflex of glaucoma" is not characteristic of the disease; it can be often seen in old eyes; the conditions that favor its appearance are a dilatation of the pupil combined with some want of perfect transparence of the aqueous humor, lens, and cornea, or of all three combined.

THE OPTIC DISC AND RETINA

CUPPING OF THE OPTIC DISC.—The typical cupping of the optic disc, on which so much reliance is placed, is a feature of established glaucoma, and is often absent in early cases. The sign may, therefore, fail us in the very class of case in which the greatest difficulty in diagnosis occurs.

The method of formation of the cup in the great majority of cases is typical and unmistakable: (1) If a case is watched from the first, a slight and subtle change is observed at the very margin of the disc; this, either around a large area or throughout the whole circumference, shows a slight depression, not sufficient to be estimated by the aid of the ophthalmoscope but quite enough to be recognized by any trained observer. (2) At a slightly later stage a faint bend can be observed in the course of the vessels as they pass over this depressed margin; by this time the whole circumference of the disc tends to be involved. (3) As time goes on, the depression in the floor of the disc increases so that it can be estimated ophthalmoscopically, and the kinking of the emerging vessels becomes increasingly accentuated.

The aberrant types of cupping are comparatively rarely met with. This is all the stronger reason for the surgeon to be on his guard and prepared to recognize them early. They are of two types: (1) The circular, central, physiologic cup, which gradually widens out toward the circumference on the papilla, and (2) the steadily enlarging physiologic cup. It is necessary to repeat that these are rare forms of cupping, and that the diagnosis of glaucoma in such cases must rest on a comprehensive survey of all the evidence that the case can afford.

PULSATION OF THE RETINAL VESSELS.—We must take the veins and arteries separately:

THE RETINAL VEINS.—It is commonly believed that retinal venous pulsation is a sign of glaucoma. This view has recently been challenged, and the writer has therefore studied the subject very closely. There can be no question that venous pulsation—and even strong venous pulsation—may be met with in eyes in which the tension is normal, and, again, that a complete absence of pulsation may be found in high tension eyes. There are, however, certain items of evidence which we must consider: (1) The exertion of a moderate amount of digital pressure on the globe will in many cases provoke a marked venous pulsation or exaggerate a pulsation already present. (2) The instillation of mydriatic drugs into the eye will, in a certain number of cases, have a precisely similar effect. (3) In a number of cases of glaucoma a very marked venous pulsation will be found present, and in not a few of these this pulsation will disappear or greatly lessen in character after the performance of a successful decompression operation. The writer's view is that when the other features of a case point toward glaucoma, the presence of a marked venous pulsation in the retina is a suggestive piece of contributory evidence. Further, the

presence of such a marked venous pulsation, whenever it is detected, should raise in the observer's mind the possibility of a threatening of glaucoma. The circulation within the eye has a marvelous power of adapting itself to altered conditions, but in the transition stage from one condition to another, the vascular system may give such evidence as we have been discussing of the strain which is being put upon it.

THE RETINAL ARTERIES.—*Spontaneous pulsation of the retinal arteries* is spoken of in the text-books as though it were a common, if not an invariable, accompaniment of glaucoma, whereas the truth is that it is a sign which is very rarely seen indeed, and then only in the course of congestive attacks of the disease, passing away as soon as the exacerbation is got under control. The writer can never recall a single instance in which he has detected it in an eye suffering from simple glaucoma or in a congestive case in the interval between two attacks. In many of our cases of acute glaucoma—and by this we mean acute exacerbations of a glaucomatous condition—the media are too hazy to allow us to see pulsation of the retinal vessels. In others we can see the vessels and note the absence of pulsation, while in yet others—and these very few in number—a distinct, and sometimes a very marked, arterial pulsation may be clearly discerned. We know that arterial pulsation means an intermittence of the even flow of blood through the arteries, as a result of the intraocular pressure having risen to such a height that it is greater than the retinal arterial pressure during the diastolic phase of the latter. In other words, it is clear evidence that the retinal circulation has become intermittent instead of continuous. Very different grades of this condition can be recognized: (1) The arterial pulse is nothing more than a flicker which affects, however, the whole course of every branch of the retinal artery on the disc and even beyond it; it can be counted against, and confirmed by the radial pulse. Here we obviously have to do with the slightest possible interference with the retinal arterial circulation; it is only at the lowest phase of diastole that the even flow of the current is interrupted. (2) The arterial pulse becomes more and more marked; the phase of blanching of the artery may be so prolonged as to equal or exceed that of its greatest fulness with blood; here the interruption of the retinal circulation is obviously becoming very serious. (3) In extreme cases the intraocular pressure has become so excessive that circulation can only be maintained at the height of systolic pressure; the phases of blanching of the vessels exceed those in which they are filled, giving almost the appearance of an aortic pulse, and justifying

the simile, used by a French writer, that the blood seems to spill in jets over the edge of the disc.

It will require but little consideration to show any thoughtful mind that the watching of this phenomenon will provide valuable clinical data to the observant ophthalmologist. So far we have been discussing the spontaneous arterial retinal pulse. We shall now turn to consider a phenomenon which can be observed not only in glaucomatous, but also in healthy eyes, and yet one which has a very definite clinical significance.

The Induced Pulsation of the Retinal Arteries.—It is well known that by the exertion of digital pressure on the globe of the eye we can produce a pulse in the retinal arteries. This pulse follows closely the characters described in dealing with the spontaneous arterial pulse: With mild pressure the phase of interruption is extremely short. As the pressure is increased it lengthens until it exceeds the period of onward flow. Finally, if the pressure is made still greater—and it is questionable whether we have a right to employ such a means—the whole circulation through the eye is stopped, and the current through the retinal arteries definitely ceases.

The real value of these observations lies in the fact that, as the intraocular pressure rises, the amount of digital pressure necessary to produce a retinal arterial pulse tends to become less and less; consequently, when we can evoke an arterial pulse by light pressure upon the eye we are led to the conclusion that the intraocular pressure has risen until it is nearly equal to the diastolic arterial pressure. The word “tends” has been advisedly and deliberately used, and for the following reason: There are cases of undoubted glaucoma in which quite considerable pressure is required before the arterial pulse can be elicited, and there are cases in which no rise whatever of intraocular pressure has taken place, in which the diastolic pulse can be very easily produced by light pressure; this latter occurrence is most often met with in young eyes. It is obvious that in the former class of cases, the arterial pressure has risen *pari passu* with the rise in intraocular pressure—one more instance of the compensatory mechanism which is to be found in the eye, and whose working we understand so little. In the latter class we appear to have to do with a condition in which the normal diastolic arterial pressure is unusually low.

PALLOR OF THE DISC.—This pallor may be found under two quite different conditions: (1) It is seen in some cases of glaucoma at a very early stage, and is then presumably due to a constriction of the

vascular supply as a result of increased ocular pressure. That this should be so will surprise no one who is familiar with the very marked blanching of the disc which is produced by digital pressure. It has been claimed that it may be a very early and very suggestive sign, in the absence of other evidence, of an increase in intraocular pressure. This is certainly a point which should be kept carefully in mind, for in no disease is it truer that every shred of evidence helps. (2) It is met with as a marked, and often as an unmistakable, feature of established glaucoma. Its interest here lies not so much in establishing the diagnosis of the disease—which by this time has usually already been made without difficulty—but in furnishing a criterion as to the probable prognosis for vision, if and after a successful decompression operation has been performed. A similar relative pallor of the retina may sometimes be observed. This is, however, very difficult to appreciate, and a more significant sign is therefore to be found in a slight relative decrease of the diameters of the arteries, as compared with those of the veins.

A dragging over of the retinal vessels in a bundle toward the nasal side of the fundus is a not uncommon phenomenon in late cases of glaucoma, and the appearance presented is very striking. Even when there are considerable opacities in the media of the eye, the bundle of red lines running inward and the contrasting dead whiteness of the disc in every other direction furnish a picture which is unmistakable. This dragging over of the vessels is probably always associated with some measure of posterior staphyloma.

THE SIZE OF THE GLOBE

According to Priestley Smith, the average horizontal diameter of the cornea is 11.1 mm. in the glaucomatous as against 11.6 mm. in healthy eyes. The importance of this fact lies in the help it gives us, when patients with a glaucomatous family history consult us as to the condition of their own eyes, being made nervous by the fear that they may have inherited the disease. Should we find that they have a low corneal diameter we would be justified in recommending them to keep themselves under periodic observation, and to report the appearance of any suspicious symptoms of the disease. On the other hand, the observation of a large corneal diameter would be a distinct factor in enabling us to give a good prognosis. The writer has been extremely dissatisfied with the devices employed for making these measurements; they seem to him far too rough and ready for their

purpose. If it is important to have the information, that information should be as *exact* as it is possible to make it. This is the principle on which all our examinations of the glaucoma patient should be conducted if the opinions we give are to be really reliable. The requisites for a perfect instrument of the kind are: (1) A head-rest must be provided, so that the patient may be absolutely steady. (2) The instrument itself must be rigidly fixed on a firm stand. (3) The observed eye must have a definite point of fixation, so that it may not make the least move during the examination. (4) The scale and the eye must be sufficiently magnified to enable the observer to measure accurately in tenths and preferably in twentieths of a millimeter. Messrs. Zeiss are constructing such an instrument especially for the writer, which will give a magnification of eight diameters. This can be substituted for their corneal microscope on the standard adjustment stand of that instrument, and will be focused by a rack and pinion movement.

SUBJECTIVE PHENOMENA

PAIN.—Speaking broadly, pain is here, as elsewhere, a measure of the congestion present. A simple glaucoma may run its whole course without one moment of pain in the eye. Again, the early slight attacks of subacute glaucoma may be attended only by mild feelings of discomfort or of ocular pressure. On the other hand, the acute disease is marked by great suffering. Our principal interest in the subject lies in the fact that the trouble is not always referred to the eye; the patient and even his medical adviser may make the mistake of thinking that the cause of the trouble is in the teeth, the ears, or the nose, since the ‘neuralgia’ complained of appears to start from one of those organs. Again, as we well know, the headache, vomiting, and pyrexia may lead to the erroneous and mischievous diagnosis of that refuge of incompetence, “the bilious headache.” The taking of a meal or the obtaining of sleep may greatly alleviate the symptoms; such drugs as phenacetin and antipyrin are useless, while the bold instillation of miotics acts like a charm. All these are points that help the careful surgeon to a right appreciation of the cause of a patient’s suffering.

LACRIMATION.—According to Morax, various reflex troubles may suggest the commencement of glaucoma; the most suspicious of these is intermittent lacrimation, unconnected with wind or other provocative agency.

NAUSEA AND VOMITING.—Morax states that, even if the glaucoma is untreated, the actual vomiting subsides within twenty-four or at most forty-eight hours, while the sensation of nausea may persist for several weeks. He has met with patients suffering from subacute attacks, in whom the feeling of nausea was the *only* warning sign of a fresh rise in intraocular pressure.

PHOTOPSIÆ are not unknown in the early stages of congestive glaucoma. They may occur as flashes of light, as the impression of a ball of fire which rolls across the field of vision, as sudden, sharp, flash-like spots of light, or as a continuous luminous glow, lasting from seconds to minutes or even longer. The patient is most apt to suffer from these photopsiæ when he is tired and when he first gets into bed at night. It must be remembered that like symptoms may be observed under any conditions in which the retina is irritated, dragged upon, or otherwise interfered with. Nor must we forget that similar phenomena, but of central origin, are met with in neurasthenic patients suffering from errors of refraction, from migraine, and from other nervous troubles. Rest tends to relieve photopsiæ of retinal origin, while those due to cerebral trouble are often worst at night.

RAINBOWS ROUND LIGHTS.—These have already been discussed.

A DIMINUTION OF VISUAL ACUITY is a frequent, if not a constant, sign of glaucoma; its causes may be classified into (1) medial and (2) neuroretinal. The former include (a) interference with the refractile power of the cornea, owing to overstretching of that membrane; (b) corneal edema; (c) deposits on the back of the cornea; (d) degenerative and other permanent changes in the cornea; (e) turbidity of the aqueous; (f) opacities in the lens; (g) deposits of various kinds on the surface of the lens capsule; and (h) vitreous opacities. (2) Neuroretinal causes include pressure on, and overstretching of, the bundles of the optic nerve and of the layers of the retina and (b) starvation of the nerve and retina due to interference with the blood supply as a result of intraocular pressure. The lesion may be manifested by a diminution of central vision or by an interference with the visual field. The former is practically always an evidence that a congestive element has entered into the case; otherwise good central vision is retained almost to the last. Defects in the field, on the other hand, are to be attributed to injury to the nerve fibers at the edge of the disc, reinforced by starvation of the retinal blood supply. The distinction is obviously important.

THE VISUAL FIELD

The subject of the changes in the visual field in glaucoma is so vast that it will be possible only to touch on a few of the main points of interest. The examination of a suspected glaucoma patient is very incomplete unless the fields have been carefully taken. Nor does one measurement suffice for each eye. The whole field should first be charted on a short radius (33 cm.) instrument, care being taken that the moving object is sufficiently small (not above 3 mm. in diameter for a daylight instrument); next, the central portion of the field, lying within 26° of the center, should be examined at a distance of 1 m. with a 1 mm. object. The results obtained by the two procedures are best kept on separate charts.

PERIMETRY OF THE WHOLE FIELD.—From this method we obtain certain very important indications: (1) There is a tendency for the nasal portion of the field to be affected before the temporal, and also for it to be more affected than the temporal, as the disease runs its course. Though this is not an invariable rule, it is so frequent an occurrence that it deserves to be kept carefully in mind. (2) As the disease progresses, the whole field tends to shrink from the periphery toward the center. (3) The blind spot shows distinct signs of enlargement, even on examination by the small perimeter, if the fields are carefully taken from time to time as the disease progresses. (4) Roenne's step appears on the charts (Fig. 1). This has been deliberately left till the last, not because it is the least important but because the writer desires to draw special attention to it. This sign is far too little known and appreciated. It is an evidence of the lesion of the optic nerve bundles at the edge of the disc (Fig. 2), and it therefore clearly points to the essential pathologic process which is responsible for the harm done to the optic nerve as a result of increased pressure within the eye. This does not mean that it is necessarily pathognomonic of glaucoma, for we know that other morbid conditions may damage the nerve fibers as the latter flow over the edge of the disc. Nor, again, does it imply any doubt as to the influence of the vascular factor in the damage inflicted on the optic nerve and retina. It is obvious that the nerve fibers and retinal elements, which are starved of arterial blood and engorged with venous blood, as a result of increased pressure within the eye, must be thereby placed in a condition unfavorable to the resistance of trauma. In this we see the contributory element furnished by interference with the

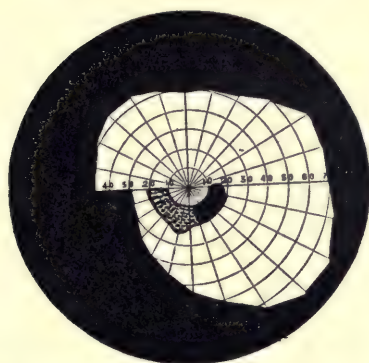


Fig. 1.—Chart from R. E. with chronic glaucoma. Bjerrum's sign is well shown. So also is Roenne's sign. The most severe lesions to the bundles of optic nerve-fibers lie at the upper temporal edge of the disc; compare with diagram in Fig. 2. Over the dotted area vision is indistinct.

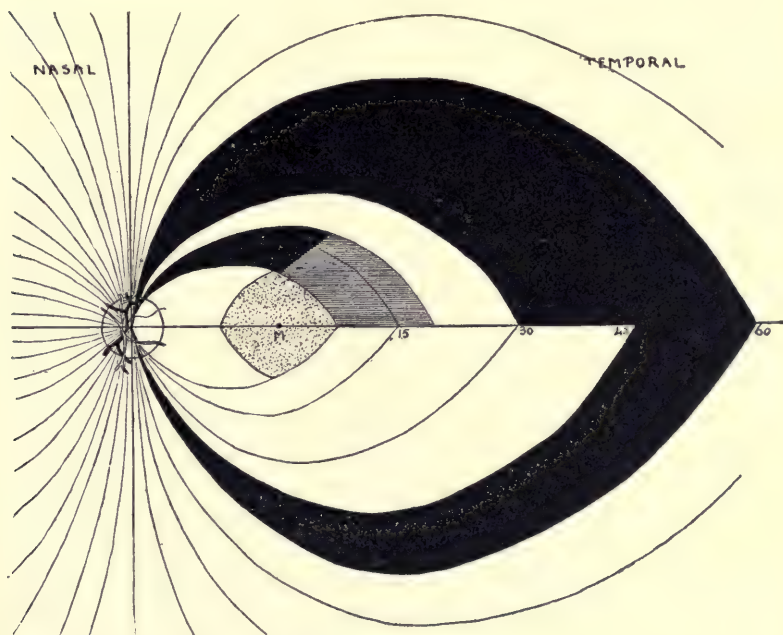


Fig. 2.—The diagram shows roughly the defects in the nerve bundles of the retina, which would correspond with the visual field defects shown in Fig. 1. The illustration is diagrammatic and only approximate.

retinal and choroidal vascular blood supply; but it cannot be too strongly emphasized that for the essential lesions of glaucoma, and for the evidence whereby we diagnose them by means of perimetry we must look to the edge of the disc and to the damage done to the nerve fibers at that area. This being so, the importance of Roenne's sign stands out large against the background of other perimetric observations. Why has it been so little appreciated, and why is it so seldom observed? The answer is: Because so much of the perimetric work in consulting rooms and other places is done against time, and sufficient leisure and trouble is not devoted to it. Moreover, as has so often been pointed out, this sign is best observed when the *circular* method of perimetry is adopted, for the simple reason that the fibers, whose damage we are studying, run in arc-like curves around the center of vision, and any lesions they sustain are more easily studied when the observed object travels along the length of the fibers instead of across them. To take eight radial measurements of a field, and then to fill in a chart by drawing lines between them is not perimetry; on the contrary, it is bad work, and is most unfair to the patient. It will not reveal the presence of a Roenne's step, nor will it give a true, or even an approximately true, idea of the state of the visual field.

One point more deserves to be remembered: The glaucomatous patient is sensitive to changes in light, and it is therefore important that periodical examinations of his field should be made as nearly as possible under the same conditions of illumination. It is difficult to obtain these by daylight in northern latitudes, and for this reason, self-lit instruments or those illuminated by artificial light are unquestionably preferable to daylight apparatus. At the same time, it is very important to know the limitations of the instrument we use; these can be learned only by constant practice. Under no circumstances must either the fixation or the traveling object be too bright. If it is so, the patient will soon become fatigued, while before this happens, the limits of his field will be exaggerated.

SCOTOMETRY OR CENTRAL PERIMETRY.—Bjerrum was the first to point out that in glaucoma we commonly meet with arc-like scotomata, which are connected with the blind spot, and which curve round the center of the field of vision to end on the horizontal raphé. Such scotomata may be met with above the horizontal raphé, or below it, or in both situations simultaneously. In the last case we get ring scotomata. He maintained that these scotomata were the expression of lesions of nerve-fiber bundles at the edge of the disc.

Roenne, working on Bjerrum's hypothesis, looked for and found the sign, which is called by his name, and which we have already spoken of in connection with perimetry of the whole field of vision.

Seidel went a step farther, and showed that very early in glaucoma, before any other sign of the disease could be certainly recognized, an enlargement of the blind spot, either upward, or downward, or both, could be detected with certainty in a number of cases. These enlargements were always described as ending in single pointed or rounded ends. From the first, it appeared to the writer that this clinical feature was inconsistent with the accepted pathology of the condition, for, if it were a question of a lesion of a number of bundles of nerve fibers selected, as it were, out of the whole mass of the nerve, it would only be reasonable to expect that the lesions they would sustain would vary widely amongst themselves, and that therefore the scotoma produced would not end in a point but in a number of points. Experiments with Bjerrum's screen and with other apparatus of a similar nature failed to confirm this suggestion, until the writer devised the scotometer which bears his name. Then the scotoma with jagged points was at once found, and has since proved to be so consistent a manifestation as to make the appearance of this phenomenon of high diagnostic value.

Three principles are involved in the make-up of this apparatus: (1) That of the circular, instead of the radial, method of examination of the field of vision, as advocated by Priestley Smith; (2) that of the magnification of the scale on which the phenomena are observed, so making the results easier to obtain and more striking to the examined eye, as advocated by Bjerrum; and (3) that of the examination of the field at intervals of 1° instead of at those of 5° or 10° as is so often done. This last was a device which suggested itself to the writer's mind as likely to bring out the jagged nature of the scotoma; if this could by any means be accomplished.

It has been pointed out by some that the writer's sign is not obtained by other forms of scotometric apparatus, and it has consequently been suggested that it is an artefact. That this argument is not a very strong one is clear from the fact that the sign in question has been obtained in glaucoma cases by a number of reliable observers, who have abundantly confirmed the writer's findings with this instrument. A little consideration will show that when dealing with glaucoma cases it would be only natural to expect more accurate results from the Elliot instrument than from an ordinary scotometer, and this

for the following reasons: (1) As has already been shown, the circular method has special advantages in dealing with scotomata which are the result of lesions to nerve fibers at the edge of the optic disc; for these fibers sweep in curves around the central area of vision to reach the horizontal raphé; and it is always much easier, both for the surgeon and for the patient if, in mapping a scotoma, we pass through its longest and not through its shortest axis. Moreover, in the author's experience, a scotoma will always be carried further, and therefore shown to the greatest advantage, if we pass from the blind in to the seeing area, instead of in the opposite direction. (2) One can explore the whole field out to the 26° circle at 1° intervals by means of 26 circles easily, accurately, and mechanically traced for us by a rotating disc; whereas to do this at the same intervals, working radially from the center would demand 360 observations and would hopelessly tire our patient. Moreover, the closely set radial lines would for the first ten or fifteen degrees be practically impossible to dissociate from one another. (3) Whatever may be the verdict of other surgeons, nothing can shake the writer's conviction that the magnification of scotomata, whether these be physiologic or pathologic, makes the patient's task enormously easier, and the surgeon's results much more accurate.

THE NORMAL BLIND SPOT, as mapped out by the new instrument, does not differ materially from the records of other instruments employed for the same purpose. This is all the more important since the field changes which it reveals in glaucoma are so very distinctive. We shall now consider these:

1. THE JAGGED ENLARGEMENT OF THE BLIND SPOT (FIG. 3).—This is so marked a feature in many cases that it cannot easily be missed by any one who works with reasonable care. Even in quite early cases the pointed enlargement of the normal physiologic scotoma may be highly suggestive of the presence of intraocular pressure.

On the other hand, if, under this test, we find the blind spot of absolutely normal size in a suspected eye, we must regard this negative evidence as of great value in suggesting that there is no rise of intraocular pressure present, or at least that any such rise that may be, or may have been, present has probably up to date done no harm.

2. DETACHED PARACENTRAL SCOTOMATA.—A certain amount, though not a great deal, has been written about scotomata in glaucomatous eyes which are found at some little distance from the blind spot. Such defects are not infrequently met with when using the author's scotometer. If such a case is followed for some time, these

defects can often be traced until they join up with the enlarged blind spot. They are obviously due to lesions of those nerve-fibers which are distributed to parts further away from the disc. Consider for a moment a bundle emerging from the optic nerve at the disc edge. It would naturally be expected that the fibers which would be earliest and most damaged would be those on the periphery of the nerve; such a lesion would be evidenced by an enlargement of the blind spot. If, however, those fibers which are distributed to the area of the

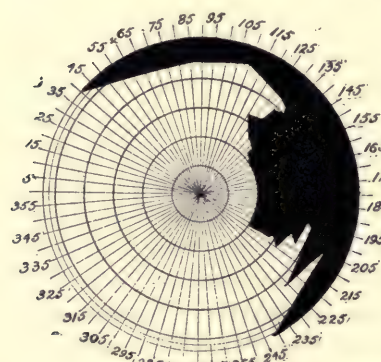


Fig. 3.—Defect mapped out by author's scotometer in an early case of glaucoma a few days before operation. Note the irregularity of the endings of the upward and downward extensions of the scotoma; these show a number of points, which presumably represent lesions of small bundles of fibers.

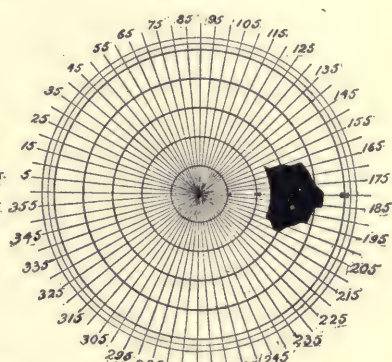


Fig. 4.—Defect in the same case as Fig. 3, ten days after relief of tension by a successful trephining. The pointed peculiarity can still be traced in this scotoma.

The test-object in both examinations was a 2 mm. disc of white blotting paper. Each circle represents 5°.

P. S.—A third test was made six weeks after the operation, and the blind spot was then found to be practically normal, having undergone a considerable further contraction.

retina, represented by that portion of the field close to the horizontal raphé, happen to be earliest damaged, we get one of these detached paracentral scotomata close to the horizontal meridian. Again, if some of the intermediate fibers are damaged, we get a scotoma in a corresponding position somewhere along the curved arc of the course of the bundle.

In explanation of these vagaries in the scotometric phenomena, the following considerations may be suggested: (a) The bundles at the periphery of the nerve are most liable to be damaged by the sharp

edge of the scleral ring over which they curve to reach the retinal surface. (b) The central bundles, destined for the more peripheral parts, are more likely to be injured by the overstretching to which they are subjected when the nerve is pushed back under pressure. (c) Variations in the anatomic arrangements of the nerve-fiber bundles themselves may possibly explain some of the discrepancies in the phenomena observed. (d) There are considerable anatomic variations in the supporting framework of the optic nerve-head, and it is highly probable that consequently the nerve-head yields differently in different eyes, and unevenly in the same eye. We are therefore justified in expecting that the injury inflicted will vary from case to case, and so will materially influence the signs of disease presented by the perimetric picture.

3. INVASIONS OF THE CENTRAL AREA OF THE FIELD.—The surgeon who employs the writer's scotometer as a routine step in the examination of his glaucoma cases, will be surprised to find how often the central area of the field is affected in eyes in which, under an ordinary superficial examination, he would not have suspected any such defect.

This is a matter of great interest and importance. The writer has seen patients whose medical men believe that they were holding their own, and whose fields, taken on an ordinary small radius perimeter, supported such a view, and yet they themselves were firmly convinced that they were losing ground, as indeed they were; careful scotometry at once showed that they had good reason for their complaints. The value of the writer's instrument in explaining such cases, in following doubtful eyes before coming to a decision as to operation, and in gauging the effect of operative or other treatment must be experienced to be appreciated.

The invasions of this area of the field tend to move steadily onward toward the obliteration of the upper, or of the lower, central field, or of both. Two clinical types are thus evolved: (1) When the scotoma is confined to either the upper or the lower field, the condition may amount to a nearly complete central hemianopia, before the patient is aware of the serious defect in his vision. This can only be the case when the opposite eye is comparatively normal. For obvious reasons the hemianopia is always superior or inferior and never lateral. (2) When the invasion of the central field takes place simultaneously above and below the horizontal meridian, the patient is more likely to detect it at an early date. The progress of the formation of a ring scotoma can be watched in such cases throughout its stages.

The diversity met with in different glaucoma fields has already been commented on, and some indication of its various causes has been furnished. We must now shortly discuss the influence of the incursion of the vascular factor on the broad features of these fields. As has already been pointed out, the field changes which are due to the mechanical effects of intraocular pressure, owe their variations to anatomic differences in the supporting framework of the nerve-head, and in the arrangement of the fibers of the nerve. If the congestive element could be excluded entirely from a case, we should get very different, but always clear-cut, perimetric pictures, and from these we would be able to speak with very little hesitation as to the nature and extent of the damage which has been inflicted on the optic nerve. This, however, is far from being the case. The element of congestion has to be reckoned with in most instances. (1) In some it dominates the whole picture, blurring all details like a fog lying across a landscape; (2) in others it modifies our findings to a considerable extent, and yet we may be quite unable to apportion the just amount of blame to the two great factors, (a) the mechanical, modified by anatomic circumstances, and (b) the congestive; (3) in yet others the vascular factor is so weak that it is with the utmost difficulty we can trace its influence. This, however, we may say truly, that, while simple glaucoma tends to produce definite and uneven curtailments of the field, the entry of the vascular factor blots out, or tends to blot out, the sensitiveness of the whole retinal area.

TONOMETRY

There are comparatively few ophthalmic surgeons—and probably none in America—who do not make abundant use of one or another form of tonometer. Even the student of to-day is trained to employ this instrument just as naturally as he does the thermometer or the binaural stethoscope. The ophthalmologist who does not do so fails in his duty to his patients and is an anachronism. Some of us use the Schiötz, some the Gradle, and some the McLean. It matters comparatively little which model we adopt so long as we keep to one and learn to understand its indications. Nor is it a matter of any great importance from the clinical point of view that the translated reading in mm. Hg may not accurately represent the intraocular pressure in the eyes measured. We cannot fix for any tonometer a point below which the recorded pressure is certainly normal, or a corresponding point above which it is supernormal, for we know that the pressure

may vary considerably in different normal eyes; what may be low for one may be normal for another, and what may be normal for a third may be high for a fourth. It is to take an altogether perverted view of the use of a tonometer to regard its readings as if they were of the nature of a scientific laboratory experiment. They are nothing of the sort, and it may be a long time before they ever become as accurate as this. On the other hand, the indications they do give us are of the greatest possible value: (1) In a very large number of cases they tell us most definitely whether the intraocular pressure is distinctly high or normal. (2) When the pressure is different in the two eyes,—and especially if there are other indications of glaucoma in the higher tension eye,—we are confronted with a very strong suggestion that a pathologic rise in pressure is present. (3) Variations in the pressure of an individual eye, occurring from time to time, afford us a most valuable criterion of the progress—favorable or otherwise—that the case is making, and of the value of the medicinal, operative, or other means that we are employing in the treatment of the case. In this connection Butler's tonometer charts deserve a mention, as they enable the surgeon to follow up his cases at a glance.

It must be made quite clear that nothing which has been written above is meant as a criticism of the effort which have been made to standardize the Schiötz tonometer. Such an aim is altogether desirable, and I would like to pay here a tribute of admiration to the splendid work done in this field by American surgeons and especially by McLean.

THE LIGHT-SENSE

There can be no question that the light-sense is profoundly affected in glaucoma. We know this well from the bitter complaints of our patients. They frequently tell us of their difficulties when they pass from light into darkness or vice versa. Moreover, bright days dazzle them, while in dull, cloudy weather they have difficulty in finding their way about. An examination of the light-sense ought, therefore, to be productive of valuable data. It has been claimed by reliable British writers that in the earlier stages of glaucoma there is a rapid reduction in light minimum sense, but only a very slight reduction in the light difference sense, while in incipient atrophy, the reverse is the case. On the other hand, French writers have obtained diametrically opposite results, finding the light difference sense the first to be attacked, and the light minimum sense to be diminished only in the

presence of optic atrophy. The writer has been very much troubled over this question: He has met with well-established cases of glaucoma in which the light-sense, tested by various instruments, does not appear to be inferior to the normal, either in minimum or in difference, and yet the patients undoubtedly suffer when their light is reduced. He suggests as a possible explanation that the central light-sense, like the central visual acuity, may long remain practically normal, while the peripheral light-sense may share the deterioration with which we are so familiar at the boundaries of the visual field. The writer would emphasize that he in no way wishes to set up his own opinions on the subject against those whose findings differ from his, and that the remarks he has now made on the subject are merely a call to other workers to persist in unravelling a tangled skein. Nor is the interest of the subject purely academic; on the contrary, the most valuable results from the point of view of the early diagnosis of the disease may well spring from such work as is now suggested. It could not fail to be of interest if members of the Congress would give their views on the best pattern of photometer for use in the examination of glaucoma patients, and if they would, at the same time, indicate the means they employ for differentiating central and peripheral acuity of vision for light.

CONCLUSION

This paper has endeavored to take up and deal with a few of the less well understood aspects of the diagnosis of glaucoma. The subject is far too vast to be treated at length and the writer is deeply conscious of the many deficiencies of his contribution. There are certain points that he would like to insist upon with the utmost emphasis:

1. In order to decide whether glaucoma is present or not, the patient should be *exhaustively* examined. This is a long, painstaking, and fatiguing business, and cannot be accomplished at one sitting, or the subject will become overtired, and the data collected will be untrustworthy. Given time, patience, and suitable equipment, any surgeon should be able to make up his mind definitely either (a) that a patient has glaucoma and requires treatment, or (b) that he has not got glaucoma, or (c) that, in the absence of definite and distinct signs of the disease, the case should be followed and watched until an unhesitating opinion is arrived at. No means should be neglected to make the diagnosis as precise as possible; the history should be taken; a routine inspection of the eye should be made in a good light without

any form of apparatus, and later an examination by oblique illumination with the aid of a corneal loupe; the refraction should be carefully estimated; the corneal diameter and the depth of the chamber should be measured; further details should be sought for with the corneal microscope, the field being illuminated by a Gullstrand lamp; the possibilities of ophthalmoscopy, perimetry, scotometry, tonometry, and photometry should be exploited to the full; halos, if present, should be measured. Then, if the surgeon, having thus made the most of the means at his disposal, is still in doubt, he has little, if anything, to lose by watchful waiting.

2. The second point is the complement of the first: A diagnosis of glaucoma should never be made on any one sign or symptom, no matter how suggestive that may be. Such a course can never be necessary, and is not justifiable.

3. Once glaucoma has been definitely diagnosed, the patient should be very carefully watched, and *if he is going downhill*, in spite of general and therapeutic treatment, an early operation should be undertaken to reduce the intraocular pressure. The recurrence of congestive attacks is a strong indication for a decompression operation. One of the most delicate tests of such recurrences is the observation by the patient of halos around lights. If the congestive attacks are well marked, the diagnosis is obvious; it is only in the very mild subacute exacerbations of glaucoma that we need to rely on such a test.

In those cases where all evidence of congestion is absent, we must look for our indications to scotometry, perimetry, tonometry, ophthalmoscopy, the testing of central visual acuity, etc. The order is deliberate, and indicates the writer's views as to the relative importance of the methods.

Much has been said and written about an exaggerated tendency to resort to operation for glaucoma. What has impressed the author more than anything else in connection with this disease during the eight years that he has practised in Europe, has been the inclination, even of very able surgeons, to postpone a glaucoma operation if it is possible to do so. He believes that where one operation is undertaken too early, very many are put off till far too late. From the days of von Graefe and de Wecker onward, it has been a guiding surgical principle that the earlier an operation is undertaken for the relief of glaucoma the better is the prospect of success. It is the operations performed at a late stage which give us the worst results, and the later the stage the worse the prognosis.

DISCUSSION

DR. JOHN E. WEEKS (New York City): I would like to emphasize one or two points. One is in regard to the cupping of the disc. In the very early stage of glaucoma we may have a cupping beginning at one margin of the disc, that is, the bending of the vessels will be evident perhaps only at the lower margin, or perhaps at the upper margin of the disc. I have recently read a statement by a German authority claiming that all bending of the vessels at the margin of the disc indicates glaucoma, and I am inclined to agree with him in that respect. Another point is the repeated examination with the tonometer of the tension of the eyeball in all suspected cases.

DR. LUTHER C. PETER (Philadelphia): The cornerstone of Col. Elliot's carefully constructed diagnostic symptom-complex of glaucoma is found in the first clause of his summary—namely, an exhaustive examination. Two of the important signs in this disease are field studies and tonometry. They are most important because they not only are "high spots" in establishing a diagnosis, but are the factors upon which we largely determine the general management of a case.

His statement as to what should be our practice in tonometry is timely and logical. It matters little what instrument is employed in measuring approximately intra-ocular tension, provided, as he says, "we keep to one and learn to understand its indication." Imperfect though it may be, it serves to relatively determine whether the intra-ocular tension is too high for safety as compared with the other eye. When used with routine care and the skill acquired by frequent measurements, any of the instruments mentioned, and others as well, will serve a useful purpose, until an instrument of greater accuracy and better adapted for routine clinical work will be available.

In the matter of field studies, peripheral changes and central disturbances are important. Peripheral changes, especially in the nasal quadrant, are early phenomena, but, as Col. Elliot has pointed out, we are not so careful to search for these errors as we might be. This, however, is due largely to the inadequacy of the instruments at our disposal—a phase of our work which we hope the Ferree perimeter will remedy.

More *vital* even than the early peripheral changes are those found in the paracentral field, because their presence means that the process has advanced to dangerous limits, and that surgical intervention can no longer be side-stepped either by patient or surgeon. The slightest evidence of a beginning Bjerrum sign, whether it appears first at the margin of the blind spot, or above or below the point of fixation, speaks for surgical interference. The development of this sign is most interesting. In not a few cases it has its origin at the upper or lower temporal margin of the blind spot—the so-called Seidel sign, but really only a variation in the beginning of a typical Bjerrum scotoma. More frequently, in the speaker's experience, it has its beginning above or below the point of fixation between the 10th and 20th meridians. The earliest evidence of its presence may be detected by a half-degree test object for green or red, either at close range or at a meter's distance. When found in this location, it enlarges

toward the blind spot and toward the periphery—at times toward the raphé, where it may meet a similar scotoma from the opposite half of the field and from a ring scotoma. At times the blind spot may show an enlargement and at the same time the area above or below the fixation point may undergo relative or absolute changes simultaneously, with healthy retina intervening. If increased intra-ocular tension is not checked, the two areas will finally unite and the typical Bjerrum sign will appear. This sign may be present without alarming changes in the peripheral field. If not sought for assiduously, the false assurance that all is well because the peripheral field is of good size may lead to error. It is the sign which the author searches for with the most delicate test available, and it is the recovery from this sign in its relative or absolute stage that determines the efficacy of the surgical measure practised. When the scotoma is fully developed and extends to the periphery, the border lines of the destroyed nerve bundles may be watched with equal interest to determine whether the surgical measures practised are holding. Its extension is the most delicate indication for further surgical interference.

As to the best method of determining even slight enlargements of the blind spot, there is much room for argument. There can be no doubt that the methodical method of approach as practised by Col. Elliot will uncover a blind area if present—not only because he determines the outline by a circular motion but because his method is painstaking and methodical. The same area can be plotted by the more flexible hand method, which, if practised in the same methodical and accurate manner, must yield even more accurate results than those of a mechanically operated instrument. It is a matter of common knowledge that the points on the scleral edge of the disc where the retinal fibers are most apt to suffer compression are not necessarily at the extreme upper and lower boundaries but on the temporal side, involving the upper and lower quadrants. Using the blind spot, therefore, as a center of study a radial movement of the stimulus is apt to uncover the total defect quite as well as a meridional movement. Equally good results, however, can be obtained by not limiting oneself to either a meridional or radial movement, but by practising a freehand movement in all directions which will bring out the defect as it exists.

MR. E. TREACHER COLLINS (London, England): I should like to emphasize some of the points made by Col. Elliot and the last two speakers. First, with regard to the halos in glaucoma. As Col. Elliot has pointed out, they may be due to change in the lens, and I think that is an important point to remember. Fine changes of the nucleus of the lens, sometimes spoken of as sclerosis, may produce persistent colored halos around lights. I have seen a patient who had had both eyes operated on because of halos due to this condition of the lens, and as the halos persisted after the operations one eye was operated on a second time; there was no cupping of the disc, no constriction of the field, no other symptom of glaucoma. I have several patients with this condition whom I have watched for a long time, but no symptoms of glaucoma have developed.

Then it is necessary to make our patients clearly understand what we mean by "halos." I had a patient come to me some years ago and ask me to tell

him if he had glaucoma. I examined his eyes very carefully with the perimeter, looked into them with the ophthalmoscope, but found no signs of glaucoma. He then said that two years previously an ophthalmic surgeon examined him as I did and then asked him if he had ever seen "rainbows." He said yes, he had, and this surgeon then said, "Well, you have glaucoma." He was a very intelligent man and he went home and got a medical dictionary and looked up all he could find about glaucoma, frightening his whole family and himself, and for two years he had lived in trepidation. Then he came to me and I told him he had no glaucoma. Several years have now elapsed and no symptoms have occurred.

As Col. Elliot says, there may be a physiologic appearance of colored rings when the pupil dilates. I know a member of our own fraternity who frequently sees these rainbow rings, especially in the evening. I have watched him for a long time—he was very anxious about himself—but he has never developed any signs of glaucoma.

With regard to Koepf's sign, there is much to be learned. It is not present only in glaucoma. It can be seen readily with a Gullstrand slit lamp or by contact illumination.

With regard to the optic disc, I think an early symptom of glaucoma is the displacement of the retinal vessels laterally toward the nasal side. In eyes where there is definite glaucoma in one eye and in the other perhaps no marked symptoms one may see this lateral displacement of vessels towards the nasal side.

One word in regard to scotometry, and that is as to the diagnosis of whether the glaucomatous process has been checked by operative procedure. As previous speakers have said, the field may show no increased contraction, the acuity of vision may remain the same as before operation, and you are inclined to say the operation is satisfactory; but the patient says his eyesight is not so good as before. If you try scotometry you will find the scotomatous area has extended around the fixation spot.

Another symptom which these patients sometimes complain of is that they get no pleasure from reading. They may have 6/6 vision, but they do not care to read, and I think the reason is that the scotoma is spreading around the fixation spot, and that patients, although they see the word they look at, do not see the next word, so the pleasure of reading for them is gone.

DR. E. E. BLAAUW (Buffalo, N. Y.): Col. Elliot said it is a simple thing to measure the depth of the anterior chamber, but I think it is difficult to know where the limits are. The iris margin is very different; the pigment of the margin has a different size and extent; the cornea is not of the same thickness and curvature in all eyes; where are we going to take the measurement, in the periphery or the center? I do not know. The change of the cornea, which has appeared to me in many instances the beginning of high tension, may be considered the symptom which Stähli called "betauung." It is only seen when the epithelial cells form small blebs, best seen in reflected light. In my very limited experience I have thought that it is not a pathognomonic sign.

I am a little surprised that Col. Elliot did not speak of gonioscopy. This gives us a new method of attacking the anterior angle.

I agree that Koeppe's sign does not include deposits on the posterior layer of the cornea. In elderly eyes frequently pigment moves away from the pupillary iris border. Koeppe values, therefore, fine pigment deposits in the crypts.

DR. J. W. NORDENSON (Stockholm, Sweden): The discussion having also treated of the methods of examining eyes in reference to symptoms of glaucoma and Col. Elliot having requested in his paper members of the Congress to tell their ways of testing the light sense, I venture to call your attention to some methods of investigation that are practised in our clinics. In testing the light sense two different things are to be examined: the sense for light-differences and the sense of light-perception. The testing of the sense for light-difference will be dealt with later in a paper by Dr. Percival, so that solely the testing of light-perception comes in question here. The testing of this function, which nowadays is of greatest importance in examining aviators and motor drivers, is done in our clinics with an instrument, originally devised by Gullstrand and further elaborated and described by S. Lindquist of Södertälge.¹ The instrument consists of a box containing a light source of constant intensity and shut at one end by a milk-glass plate covered by a sieve with 900 holes. With the aid of two slides any number of these holes can be opened and shut according to wish. In making the test all the patients are assembled in a totally dark room where they are left for about half an hour in order to get adapted to dark. The instrument is then put up in front of a square piece of white paper of certain dimensions and the holes are opened one after another until the examined patient affirms that he can see the paper. The intensity of light coming from the paper when lighted by one hole, having once been tested, the examination gives you the amount of light required to give the patient perception of light. Simulation is avoided by repeating the test several times and by comparing the number of holes required each time. The method, which is a modification of the apparatus of Foerster, has the advantage that the examiner himself need not be adapted for dark. If it were possible to devise a method to control the fixation of the patient, it would be possible to use the instrument also for testing the sense of light-perception in different parts of the retina.

For the use of the slit lamp to measure the depth of the anterior chamber of which Dr. Blaauw spoke, an instrument has been constructed by Lindsteadt of Stockholm.² The principle of it is that the pencil coming from the slit is made astigmatic by an optic system, whereby one of the focal lines is placed on the anterior surface of the cornea, the other on the anterior surface of the crystalline. With knowledge of the power of the astigmatic system that is required to so adjust the focal lines, the depth of the anterior chamber can be determined very exactly.

MR. J. GRAY CLEGG (Manchester, England): Several points I wish to

¹ Upsala Läkareförenings förhandlingar, 1906.

² Arch. f. Augenheilkunde, vol. 80.

mention, and one is that in making a diagnosis we cannot rely on any absolute line as to the height of pressure when determining the presence of glaucoma in the particular eye under examination. One finds a considerable variation, and perhaps this is well illustrated by a case that I have had recently, an iridocyclitis with high tension, but where there was perfect peripheral vision, no scotoma, no enlargement of the blind spot, but the tension was high—over 40 mm. It was only after some months of treatment, the tension remaining high, that slight enlargement of the blind spot was found and operation was resorted to. Other cases show the signs of chronic glaucoma, although the tension is never found above normal.

In the old days my predecessor, Dr. David Little, used to be afraid of the iridectomy operation when there was a blind area near the fixation point, for fear the surgical interference might, as it were, tip the eye over the precipice; but we find that with the trephining operation we need not hold back, even though the blind area extends within two or three degrees of the central point.

Another important point is that we must not overlook the general health of the patient.

DR. GEORGE F. KEIPER (Lafayette, Ind.): I want to add the history of a patient with chronic glaucoma, who before I first saw him had lost one eye, and I have been afraid to operate on the other eye, and have been controlling him with miotics. When I drew the curtains down in the dark room he said he could see the test letters better when the room was brightly illuminated. I tried that with him a number of times, and found it to be true. I now make this test with all persons over the age of forty, and if I find they can see the letters in the dark room better with the curtains up rather than in a shaded room, my suspicions are immediately aroused, and I am inclined to think of the advice Dr. Ellett gave years ago, to use tonic drops of eserin in order to be on the guard against cases of glaucoma that may manifest themselves by any other symptoms.

DR. ARNOLD KNAPP (New York City): While we may not all agree on the form of operation, we are all united on the fact that the value of any treatment of glaucoma rests in the early diagnosis, and the most important factor in this is the examination of the paracentral area by the scotometer. Although it is many years old, the profession in general owes Col. Elliot a debt for so instructively drawing our attention to this method.

The other point I wish to mention is that among the early signs there is a curious susceptibility of the glaucomatous eye to adrenalin. In some investigations made and reported to the American Ophthalmological Society last year, I drew attention to the fact that adrenalin causes a dilatation of the pupil in glaucoma in nearly all cases. In some cases the tension was reduced, in some it remained the same, and in a few it was increased. When adrenalin was instilled into the other eye which has had no symptoms of glaucoma, a dilatation of the pupil was observed.

DR. GEORGE W. JEAN (Santa Barbara, Cal.): With regard to the loss of blood-vessels at the disc edge, which Dr. Weeks said he had recently seen mentioned in a foreign text-book, I would like to call attention to the fact

that I spoke of this sign in my text-book published in 1915. Men working under Lauber in Vienna a dozen years ago will well remember how he insisted that this sign, if present, was an absolutely pathognomonic sign of glaucoma. There are two exceptions: in congenital colobomas of the nerve head and also in those peculiar cases with a congenital hole in the disc that often goes several millimeters deep. Lauber has reported several cases of the latter, and I have seen one such case with lost vessels at the disc edge.

Of course, the sign is not always there, but with the exceptions mentioned "the short bend of a single vessel immediately at the disc edge is sufficient in every case for the diagnosis of glaucoma."

COL. R. H. ELLIOT (closing): With regard to Dr. Weeks' remarks, I have observed, as he says, over and over again, that cupping occurs first at one portion of the disc, and I think that is explained by Fuchs' recent work on the difference in the way in which the disc yields unevenly, according to the state of the distribution of the fibrous tissues in the lamina cribrosa. I am not prepared to go as far as some of the speakers who feel that a single vessel out of place means glaucoma, because I have seen cases where vessels at one part of a disc were suspicious, but when the cases were followed for a long time I could not satisfy myself that there was glaucoma present. At the same time, though, the sign always makes one suspicious.

I would like to press home what Dr. Peter said about the tonometer. The tonometer is the only accurate method of measuring the tension of the eye, and to do anything else is equivalent to putting your hand on a patient's skin and saying he has a temperature of 102°. I also agree with him that when paracentral scotoma is present a dangerous stage has been reached, and the patient should be watched; also that detached paracentral scotoma is extremely common in glaucoma.

All I claim for my instrument is that you can make a reliable clinical examination in a remarkably short time by its use. I have never put it forward as an instrument for scientific research, but it is an instrument of great delicacy and can be used rapidly.

Mr. Collins spoke of the gentleman who saw rainbows. I would like to pass this lycopodium slide around. I keep it in my dark room and say to my patient, "Have you ever seen halos?" He does not know. Then I say, "Look at the bright light through this." He looks and knows at once what we mean by a halo.

Dr. Blaauw took me up on the question of the depth of the chamber. When I wrote that I had no idea I was being so dogmatic. That was written months ago, and I would have been less assertive if I had written it now, after more experience with the Ulbrich drum. All the same, I believe it can be made a useful adjunct to diagnosis.

With regard to contact glasses, I have not had the courage yet to use contact glasses, but I am in hope of being put in the way of using them. I admit it is an important matter.

Dr. Nordenson spoke of a light-sense apparatus. I have had constructed for me by Zeiss an apparatus which I am using in my dark room and which will, I think, prove useful.

Mr. Clegg raised the important point of the general treatment of the patient. Of course the glaucomatous individual has a sick eye in a sick body, and you must examine the whole patient.

With regard to Dr. Keiper's point, the thing that strikes me more than anything else is the way in which every glaucomatous patient has deficient light sense, and yet there is difficulty in getting him to show it by any ordinary sort of apparatus. The explanation that I offer I have already given you elsewhere.

I think Dr. Knapp's test is one that we should follow very carefully.

BLOOD-PRESSURE IN THE VESSELS OF THE EYE

DR. A. MAGITOT AND DR. P. BAILLIART

Paris, France

It has long been known that a slight compression of the eyeball causes arterial pulsation in the retina. Physiology enables us easily to understand that this arterial pulsation appears when the tension of the ocular media reaches the diastolic pressure of the central artery of the retina. Similarly, we knew, that the pulsation will cease when the pressure exercised through the ocular media is greater than the systolic pressure of the central artery. It was soon discovered that, in the case of patients suffering from arterial hypertension, it was necessary to exercise on the eyeball a strong pressure to bring about the disappearance of the retinal pulsation; Bailliart (1909), then Melville Black (1911), and Deyl (1912), had drawn attention to this method of recognizing the existence of arterial hypertension.

In order to measure the arterial pressure in the central artery of the retina, it remained therefore only necessary to know the amount of force it was necessary to apply to the eyeball, to bring about the appearance and disappearance of the pulsation. Henderson had, for this purpose, constructed, as early as 1914, an apparatus which calculated in millimeters of mercury the pressure which, exerted on the exterior surface of the eyeball, was sufficient to bring about the appearance of the diastolic pulsation. Henderson thought, which is not altogether incorrect, that by adding the ocular tension to the figure given by his apparatus, he would get the diastolic pressure of the central artery.

Bailliart, in 1917, suggested a method for determining the figures of the diastolic and systolic pressures of the central artery. By

means of a special dynamometer, graduated in grams of water so that it might be easily verified on any scales, a pressure is exerted on the eye in the region of the insertion of the rectus externus, until the operator notes the appearance and then the disappearance of the retinal pulsation. Two successive readings will thus give the amount of force which it is necessary to exert to balance the diastolic and then the systolic pressure of the central artery. But the thing that it is then important to know is, how the primitive ocular tension has been modified by these dynamometric pressures, for, finally, it is by its intermediary that we act on the vessels of the eye.

Henderson has had the idea of measuring in millimeters Hg the pressure exerted on the eyeball and of adding to it the ocular tension determined by means of Schiötz's tonometer; but in this method there is a double cause of error, first, from a physical point of view, two successive pressures do not add up together, and on the other hand, the pressure transmitted by a liquid is proportional to the surface compressed. Here is, however, another method: In a dark room, the patient being placed in a recumbent position, the operator causes the appearance and disappearance of the pulsation and notes the weight in grams necessary to obtain these two results. Then Schiötz's tonometer is applied to the eye while at the same time by means of the dynamometer a pressure is exerted equal to that which was found necessary a few moments before to produce the appearance and disappearance of the pulsation. The tonometer weighted with its heaviest weight will indicate approximately in millimeters Hg the pressure exerted on the vessels of the retina. It is evident that the greater the pressure exerted on the eyeball the greater will be the ocular tension.

But this method complicates the operation and is only possible with exceptionally docile subjects. It is for this reason that we undertook to study experimentally, on an animal, the modifications of the intraocular tension under different pressures and to draw up a chart. But to be of use clinically the knowledge of these modifications of the ophthalmotonus caused by pressure exerted on a healthy or diseased eye, must apply not only to a particular case, but to a whole series of figures of initial tensions. The method would indeed be singularly restricted, if we only knew the tensions produced by a pressure of 10, 20, 40 or 80 grams on an eyeball the initial ophthalmotonus of which would be uniformly 20 mm. Hg. It is indispensable to acquire a knowledge of the effect produced on different initial

ophthalmotonus, for we must evidently suppose that the same pressure of 20 grams will produce a different tension in an eye of which the primitive ophthalmotonus was 15 mm. Hg and in another of which the ophthalmotonus would be 30 mm. Hg.

The drawing up of such a chart was possible only by experiment. We carried out researches on cats anesthetized with chloralose, this product being easier to manipulate than curare and causing only a slight modification in the general blood-pressure, on condition that the animal is not allowed to get cold. We have also made use of a mercury manometer invented by one of us and of which the manipulation appears to us to be easier than of Wessely's or of other instruments derived from Schulten's.

The ocular tension of the cat oscillates between 15 and 25 mm. Hg according to conditions. It was, therefore, relatively easy to establish the figures of ophthalmotonus starting from these initial tensions. But it was necessary to obtain greater tensions and others still weaker. We were able to produce higher starting figures by practising subconjunctival injections of NaCl. We were thus able to obtain quite easily tensions of 40 or 45 mm. Hg, rarely more.

In order to operate on lower ocular tensions than 15 mm. Hg we had recourse to a deeper narcosis in order to act upon the general blood-pressure. But we were unable to obtain less than 10 mm. Hg which in the cat, as in man, is the immediate postmortem tension.

We chose the cat in preference to the rabbit (which is, however, an easier animal to handle) on account of its ocular nervous system, which is very similar to that of man. Yet both of them possess a much larger anterior chamber and we do not know whether the general elasticity and resistance are identical with those of the human eye.

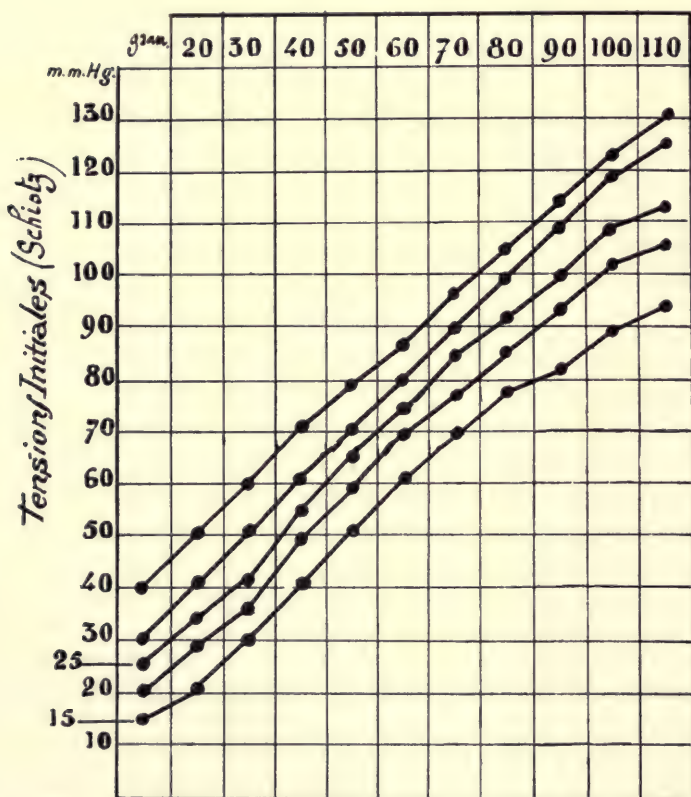
We have avoided producing modifications of the tonus by injection or aspiration of the intraocular liquid as these means give erroneous figures for the two following reasons:

1. On account of the resorption.
2. On account of the intraocular vasodilatation which, very probably, takes place after a few seconds. As to the pressures, they are made by means of Bailliar's dynamometer, which is either held in the hand or immovably fixed in a ratchet-support.

The chart on next page will avoid any useless descriptions. It gives us the figures for pressures applied horizontally (as on a patient sitting or reclining) on eyeballs of which the initial tensions vary from 10

mm. to 50 mm. Hg. It will be noted that the different lines starting from different initial tensions are remarkable for their parallelism and also for being nearly straight. It results from this that the chart can be completed for all the initial pressures and for all pressures applied.

Pesées horizontales (grammes)



It will be seen that with this chart the determination of diastolic or systolic arterial pressure in the branches of the central artery becomes easy.

We first measure with Schiötz's tonometer the initial ocular tension, then we seek what dynamometric pressure will bring about the ap-

pearance and later the disappearance of the arterial pulse. We then refer to the chart and we find in millimeters Hg the required figures. In an eye of normal tension in a subject whose arterial pressure is normal, the arterial retinal pressure is about 35 mm. for the diastolic and 70 mm. for the systolic.

If the retinal circulation has a primordial importance since it insures the visual function, the choroidal circulation also plays a considerable part; the part it takes in the modifications of the ophthalmotonus is well known. It is moreover possible by studying the effects produced by compression of the eye to obtain on the uveal circulation notes similar to those obtained on the retinal circulation.

Sometimes, particularly in extreme myopia, it is possible to perceive through the retina the choroidal vessels; but we can but ill distinguish the arteries from the veins and on the other hand these vessels never show, except in certain exceptional cases, either spontaneous or forced pulsations. When considerable pressure is applied, it is even difficult to tell at what precise moment they are emptied, and, even if we did know it, this would have little value since we cannot distinguish the small arteries from the small veins.

Consequently, the direct examination and measuring of the arterial pressure in the choroidal vessels is not yet possible.

In a recent work G. Leplat has shown that it was possible under certain favorable conditions to study "*de visu*" the blood-pressure in the arteries of the dog's iris by making use of the process we have described. The importance of this fact is truly considerable; this process enables us to acquire for the first time some ideas on the circulation of the uvea of which the iris is the sole portion which is clearly visible.

In man, unfortunately, the iris circulation, like the choroidal circulation, does not lend itself to these investigations, and, however great was our desire, we were unable (even with the magnification of Czapske's loupe) to observe any iris pulsation. It may be that we should see in this fact a confirmation of Thomson Henderson's opinion that the arterial circle of the iris in man is in reality a venous circle.

The dog, which G. Leplat used for his researches, possesses in the periphery of the iris, particularly on the side toward the nose, veritable arterial trunks of which the beatings can be seen with a magnifying glass of low power or even under good conditions of lighting, with the naked eye. The observation of the pulsatile arterial reac-

tions is thus very easy and enables us to study, as our Belgian colleague has done so well, the pressure in the vessels of the iris. On our side we have undertaken a series of researches for the purpose of studying comparatively the iris circulation and the retinal circulation; and we have made use of the cat. The dog, which is a good subject for the vessels of the iris, appeared to us to be of no service for the study of the vessels of the retina; for its central vessels when they reach the disc are already divided into fine branches surrounded by neuroglia tissue. This disposition does not allow the beating to attain the amplitude desirable.

The retinal vessels of the cat, apart from their being disposed on the periphery and not in the center of the disc, and their ciliary origin, resemble on the contrary fairly exactly the vessels of the human retina. At the limit of the dark gray disc we see three or four arteries each with a vein running by its side. The artificial beats which we provoke must always be sought for in the immediate circumference of the disc. They are, moreover, very easy to observe; when the appropriate pressure is applied to the eyeball, three or four small arteries can be seen beating simultaneously.

The vessels of the iris are *per contra* rather less easy to observe than those of the dog. In order to see them well, it is necessary to make use of Czapski's magnifying glass, and to examine the nasal extremity of the horizontal meridian; a voluminous vein (particularly if one looks very closely at the root of the iris, before its bifurcation) will be seen lifting up the trabecular tissue; at this spot we recognize the existence of spontaneous arterial movements which pressure on the eyeball exaggerates and transforms into veritable pulsations and finally suppresses completely. If the retinal pulsations of the cat are always easy to see, it must be added that the pulsations of the vessels of the iris are sometimes less so.

The comparative study of these two pulsatile phenomena has enabled us to remark that in the eye of the cat the pressure in the arteries of the iris and of the retina is practically the same; if in the average case it has been necessary to apply somewhat stronger pressures to study the pulsation in the iris than in the retina, this is due, no doubt, on the one hand to the greater difficulty of observation in the iris. We can give as an average in the cat for the arteries of the iris and the retina 45 to 100 mm. Hg, which figures are slightly higher than those in man.

These experiments on animals have led us to recognize (which we consider to be quite probable) that the systems of blood-vessels of the retina and the uvea, so similar to each other in many ways, have in their normal state an identical working pressure.

We have since had fresh confirmation of this in a work of Vossius. This author had occasion to examine with the corneal microscope a filament of an iris in a persistent pupillary membrane. On applying dynamometric pressure to the eyeball he noted that circulation was arrested when the ocular tension reached 70 mm. Hg. This figure (70 mm. Hg) is also that which we found for the normal systolic pressure.

The study of the effects of graduated and known pressures on the eyeball has also led us to other results. We have been able to observe a constant phenomenon which can be formulated as follows:

1. From an initial tension of 15 mm. there exists always a lowering of the ocular tension after each application of the tonometer, however correctly the application be made. Example: take an eyeball of which the tension is 20 mm. Hg; submit it for a duration of three seconds to a pressure of 30 gram. We shall find immediately after that the ophthalmotonus is no longer 20 mm. Hg, but only 15 mm.

2. This phenomenon no longer exists when the same pressures are applied to eyeballs possessing only a tonus of 10 to 12 mm. Hg.

3. This phenomenon is exaggerated when the pressure is applied to eyeballs in which the tension has been raised artificially (in our experiments by means of subconjunctival injections of hypertonic serum).

These modifications of the ocular tonus under the influence of slight pressures applied to the eyeball had already been noted in tonometric experimentation. This observation had already been made clinically in 1911 by Polak Van Gelder. Using Schiötz's tonometer, he remarked that in normal individuals the repeated applications of this instrument at intervals of 3 or 5 seconds always furnished figures lower than the first ones. If we translate into grams the pressure exerted on the eyeball by the tonometer loaded with these weights of 5 or of 7 grams, we see that it varies from 21 to 25 grams. In Polak Van Gelder's tables we note that the difference between the ocular tensions before and after this tonometric pressure is about 10 mm. Hg.

This fact is also quite clear when dynamometric pressures are applied to the eyeball in order to measure the arterial retinal pressure.

If, when by means of a given pressure we have managed to efface the last arterial pulsation, this pressure is maintained for a few seconds, it will be seen that the arterial pulsation reappears very rapidly; to suppress it, greater pressure must be applied; if this pressure is maintained the pulsations reappear. Thus the ocular tension diminishes immediately and constantly under the influence of dynamometric pressure. This fact must be noted as it explains why beginners, who proceed by feeling their way, always find very high figures in their attempts to determine the arterial retinal pressure.

This lowering of tension under the influence of pressure is, moreover, very transitory: to the depression there succeeds even a slight ulterior elevation which lasts a very short time.

How can we explain this diminution of the ocular tension under the influence of ocular pressure? Evidently by the issuing of a part of its liquid contents. The supporters of the continuous stream of the aqueous humor explain it by a more active filtration of this liquid which is driven out of the eyeball by the mechanical effect of the compression. For our part, we believe that the question is not so simple. Remembering the modifications of the ocular tonus under all the influences which can act upon the circulatory system of the choroid, the fall of the ophthalmotonus after ligature of the ciliary arteries or of the carotid, after stimulation of the "sympathetic," its rise which follows inhalation of amyliis nitris, after division of the sympathetic, we remain convinced that the compression of the eyeball acts by emptying the choroidal system, and that this mechanical expansion of the external coat of the eye brings about the fall of the ocular tonus, which rises again when little by little the blood fills once more the exceedingly rich vascular system of the uvea.

PLASTIC OPERATIONS ABOUT THE EYE

SUGGESTIONS FOR SEVERAL IMPORTANT CONDITIONS

DR. JOHN M. WHEELER

New York City

In considering the subject of plastic surgery of the structures about the eye, I shall limit myself to some of the more important features of operations which have been developed in my experience, while attempting to determine the best methods of correcting deformities in the eye region. In my opinion surgery of the eyelids, eyebrows and orbital region should be performed by ophthalmologists, and not by those who have no special knowledge of surgery of the eye region. General surgeons are uninformed on the peculiar features and requirements here, and they are not trained in the refinements essential to success in plastic eye surgery.

Plastic eye work enriches the field of ophthalmic surgery and broadens the scope of the ophthalmologist without interfering with the special skill which he must develop in order to perform successfully the important operations on the eyeball proper; and certainly nothing should be allowed to detract from the high importance of the skill and refinement and judgment called for in handling such conditions as cataract and glaucoma.

In studying and applying methods for handling cases of deformity about the eyes I have always had in mind these two things. First, to avoid adding new deformities through my surgery. Second, to develop procedures which are as simple as possible and which are appropriate for general adoption by trained eye surgeons.

In order to make satisfactory comparisons of the relative merits of different sorts of grafts in corrective surface work of the eyelids and thereabouts, I have used different types of grafts and different methods on the same patient. For instance, one patient who needed more than one correction received a true skin flap from the inner aspect of the arm, a free graft of true skin from one temple, and a pedunculated flap from the other temple. Another received epidermic grafts from the thigh, and true skin from an upper lid. Another, a pedunculated

flap from the temple, a free graft from the arm and a free graft from the upper eyelid, and so on. Thus, by making different sorts of combinations, it was possible to study results in an unprejudiced and convincing way.

CICATRICAL ECTROPION

SIMPLE CICATRICAL ECTROPION.—This is the ordinary variety that we have to deal with most commonly, in which the lower eyelid, or more rarely the upper, is pulled away from the globe by scar tissue, but in which the lids otherwise have suffered little. In most cases of traumatism resulting in ectropion, only a single eyelid is involved, and the lower lid suffers more frequently than the upper. For simple ectropion, by far the most satisfactory correction can be obtained by means of an inlay of true skin from one of the upper lids. It is surprising how seldom one or the other upper lid will fail to provide ample skin for grafting. So for most cases of cicatricial ectropion I employ detached upper lid dermis. A match in appearance is assured without the creation of an additional deformity. There need be no fear for the upper lid on account of removal of the skin. A fusiform flap of true skin 50 mm. long and 25 mm. wide can be taken safely from most any normal upper eyelid, and some could furnish much greater width. In one young adult I have taken, at intervals of a few months, three separate grafts from the left upper eyelid without lagophthalmos or appreciable deformity. The stretching of this skin tissue and readjustment to the normal appearance is remarkable. A description of this operation was published in 1921.¹

Technic of Operation.—The primary incision should be parallel to the distorted lid margin. Cicatricial tissue should be so thoroughly and painstakingly removed that there will be no tendency of the lid to evert, and scarcely more than normal resistance to upward traction at the lid margin (Fig. 1). In making the preparatory dissection, the tissues should not be handled roughly. Artery clamps should not be used unnecessarily and no ligatures should be used on bleeding vessels.

Either two or three firm adhesions between the upper and lower lids are made by dissecting off epithelium at corresponding positions on the upper and lower lid margins, and carrying mattress sutures through the little raw surfaces. They are passed through small plates of rubber (cut from sterile rubber tubing) overlying the skin of both upper and lower lids near their margins (Fig. 2). These sutures are

¹ Jour. Amer. Med. Assn., July 19.

tied snugly to insure firm apposition of the opposing raw surfaces. Union of these raw surfaces causes the formation of adhesions for the support of the lower lid. Later, these adhesions stretch somewhat and are covered with a growth of epithelium. This stretching allows of very slight separation of the lids, so that the patient can see through the palpebral fissure, a thing to be encouraged, as elevation of the upper lid stretches the lower. With this in view, it is well to avoid making an adhesion directly in front of the pupil.

Removal of the True Skin Graft from the Upper Lid.—This is very

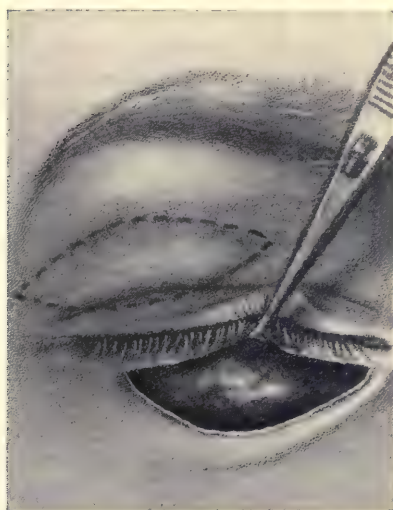


Fig. 1.—Simple cicatricial ectropion of left lower eyelid. Dissection in lower lid completed and graft outlined in upper lid.

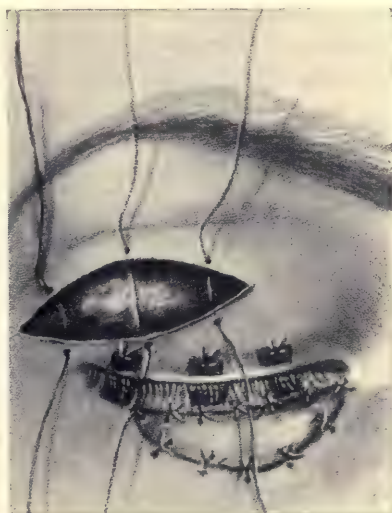


Fig. 2.—True skin graft from upper lid sutured in position in lower lid.

simple as compared with the dissection of a dermic flap from the arm, thigh or elsewhere. Almost no allowance need be made for contraction. In fact, a graft exactly the dimensions of the raw surface to be covered will answer. This graft can be taken from either upper lid, but ordinarily would be taken from the ipsilateral lid. In removing the graft, I do not use a grasping forceps of any kind for fear of bruising the tissue cells. Outlining incisions are made through the dermis of the upper lid. A fusiform or semilunar shaped design is convenient to take. A cataract knife is slid under the skin from the lower incision

to the upper. The knife is carried by careful sliding motions nearly to one end of the graft. Then it is turned about and carried to the other end, freeing it. The piece of skin is picked up with the fingers and set free.

By placing the graft, epithelial surface downward, on a pad wet with warm (body temperature) normal salt solution, any fragments of subcutaneous tissue can be quickly snipped off with the scissors. This is not a tiresome process as it is in the case of an arm graft. Fine silk sutures (No. 1 twisted) impregnated with paraffin are used to stitch the graft in position. The ends should be secured first, and then the margins should have as many sutures as may be needed to hold them in place. No undermining is necessary in order to bring the wound edges together on the upper lid. The raw surface is easily closed over by fine silk sutures, and there need be no fear of subsequent opening of this wound.

Dressing.—The graft should be covered with rubber tissue, having the slightest smear of sterile petrolatum. I prefer to put the tissue on in two layers with the grains running at right angles, to guard against the possibility of perforations through separation of the tissue fibers. The rubber tissue will prevent the skin graft from getting dry. If a covering other than rubber tissue is chosen, it should be of smooth surface, pliable and without perforations. Over the tissue, gauze fluff is packed and secured firmly by adhesive plaster, and then by pressure bandage, which in turn should be secured by adhesive plaster. It is well to put a separate dressing over the other eye, to be left for two or three days, and then to be cut down without disturbing the main dressing and bandage. This should be left for six days, at the end of which time it is removed with the utmost care, and the graft painstakingly cleansed with damp (not wet) boric acid sponges, and all sutures taken out. At the first dressing the grafted skin will appear pink, not anemic like a graft from the arm. The outlines of the beautiful inlay are hardly seen even at the first dressing. Rubber tissue, gauze dressing and bandage, changed every two days, are continued for another week; then dressing is no longer necessary, and the graft is kept smeared with a little sterile petrolatum.

About three weeks after operation it is well to start massage with petrolatum. This is kept up daily for several weeks. The adhesions between the lids should remain for at least three months. They should be left until all tendency to malposition of the lid has passed. They are then cut with the scissors and no deformity results from them.

SEVERE ECTROPION DUE TO BURNS.—Occasionally we have to deal with extreme ectropion of all the lids associated with facial burns, and large loss of the cilia and brows. Usually the outer canthus is pulled well down out of position and there may be epicanthal folds. In such cases it may not be feasible to implant true skin grafts, and it is convenient to resort to epidermis. Fortunately, in these bad burn cases epidermis matches the scarred tissue of the face about as well as true skin from a distant part, and it is possible to cover as large areas as necessary.

Operation.—Usually general anesthesia is called for in the severe

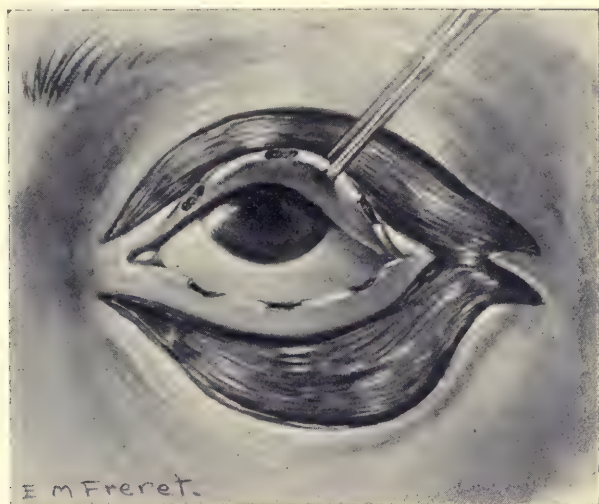


Fig. 3.—Operation for severe cicatricial ectropion. Dissection for replacement of eyelids completed. Upper lid held by forceps to show areas denuded in margin for intermarginal adhesions.

cases. After the preparatory dissection of the eyelids, there is a large exposure for the reception of the epidermic graft. Both upper and lower eyelids are repaired at the same sitting. Fig. 3 shows at the lid margins the little areas which have been denuded of epithelium in preparation for the adhesions to hold the lid margins together. These adhesions are important in the correction of all ectropion cases, but especially so in the severe cases where large denuded areas have to be covered. After passing the sutures to secure apposition of the raw areas of the margins, a single large piece of epidermis from the outer

aspect of the thigh is placed over the lids, overlapping the margins of the denuded areas all around. For this purpose a graft with an area of 9 to 12 square inches may be needed. (It is taken as for restoration of the socket.) No sutures are used but the pressure of a secure dressing and bandage are relied on to hold the graft firmly in place. After placing the skin in position a slit should be made in it just in front of the palpebral fissure to provide for drainage of conjunctival secretion. A slit is also made in the rubber tissue which is placed over the graft (Fig. 4). Dressing and after-care are the same as described for the

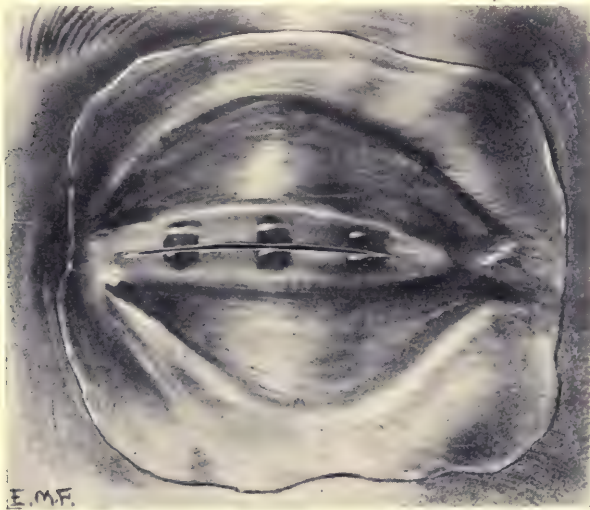


Fig. 4.—Eyelids in position for reception of epidermic graft. Sutures have been passed through denuded areas in lid margins and tied over little plates of rubber. A single epidermic graft has been placed over denuded areas in lids. Slit in graft in front of palpebral fissure.

dermic graft. In cases with bad facial scars, especially if there is a tendency to keloid formation, it may be wise to leave the interpalpebral adhesions for as long as a year, and it may be necessary to graft a second time if late contraction of facial scar tissue pulls the palpebral fissure out of place. When the work is completed the external canthus should be on a level with the internal. Let me suggest an important precaution in regard to the preparatory dissection of the lids. It is permissible for the denuded areas of the upper and lower lids to communicate at the temporal side of the palpebral fissure, but it is a mis-

take for the surgeon to establish a communication at the nasal side, as a fold will form during the healing process and make a cicatricial epicanthus. This gives a cosmetic blemish for which we have no satisfactory operation.

If the brows have not been destroyed, it may be worth while to graft from them for cilia lines, before the interpalpebral adhesions are severed. Otherwise artificial lashes may be resorted to.

An important question which is apt to confront us in connection with the severe burn cases, is whether to operate early, before the facial burns are healed, or to wait until cicatricial contraction has progressed. I should operate as soon as the physical condition of the patient will allow, even if it means operating again later on. This judgment is based on the importance of protection for the cornea by the lids, which will be held by the adhesions between their margins.

In ectropion I now find use for pedunculated flaps only in those cases where there has been loss of bony tissue with a consequent depression to be filled in. Here thickness is called for. A flap can be turned down from the temple or forehead by the well-known methods of procedure, and the thickness can be gauged to suit the needs of the case; that is, where the depression is greatest the flap can be made thickest.

RESTORATION OF BROW

Dislike for making new deformities and desire for simplicity have kept me from turning pedicled flaps from the hairy scalp or elsewhere in attempts to bring about the formation of new brows. I have used only free grafts. Loss of the brow is usually not complete, and rather satisfactory patch work can be done in some cases by taking from one part of the brow to fill out another, or by taking from one brow to help out its fellow.

If one brow is intact and the other partially or wholly destroyed, the intact brow can be divided longitudinally and the lower part transplanted as a detached graft to the other side. In order to have the hairs of the transplanted graft slant toward the temple, it must be turned upside down. This splitting procedure is not altogether satisfactory, as the resulting brows are usually noticeably narrow, and the direction of the hairs in the graft is not ideal. Moreover, some of the follicles are liable to be destroyed in the process. The reason for selecting the lower part of the brow for transplantation is that the skin tissue is much thinner below than elsewhere in the brow and the hair-follicles are shorter. This is true to such an extent that a graft includ-

ing the hairs of the lower part of the brow is wedge-shaped on cross-section, with the apex of the wedge downward.

Possibly the most satisfactory way of grafting for an entire brow or a large part of one is to remove a flap of scalp from the occipital region of the desired size and shape and place it in position. The hair-follicles are usually closely placed in the middle of the occipital region, and they are well slanted. Moreover, no visible scar results. One objection to the scalp graft is that the color of the hair may not accurately match the color of the fellow brow. Another objection is that a part of the graft may be lost. The reason for this is that the scalp tissue is thick, and the graft cannot be made thin without injuring the hair-follicles, which are long enough to go entirely through the scalp skin. One cannot transplant thick detached grafts with confidence, as is possible with thin skin grafts.

A few suggestions as to the technic of implanting the scalp graft may be offered. Novocain infiltration anesthesia is satisfactory. In preparing the bed for the reception of the graft, a curved incision through the thick supraorbital skin is made in the proper position and of the proper length. Usually the skin is on the stretch on account of the presence of scar tissue and the wound gaps in admirable fashion, so that it may not be necessary to remove any skin in order to get a good exposure of the subcutaneous tissue. The thick scalp graft will not set into the depression made by the skin incision, but will project and its life will be endangered. So a longitudinal incision should be carried along the middle of the exposed area down to the periosteum or near it. Usually it is possible to avoid the supraorbital nerve and vessels. Regeneration of the nerve can be expected, however, if it is cut.

In preparation for removal of the graft the scalp area should be shaved, cleansed with alcohol and ether, and painted with iodine. The graft needed is usually rather long and pointed. The curved incisions for its removal should slant toward each other, so that a cross section of the graft is like a truncated wedge with base at the surface. This will fit deeply into the depressed exposed area in the brow region, as the sides of this depression will be "banked." In taking a scalp flap there is inevitable destruction of some of the hair-follicles, but with a complete take or nearly that a good mat of hair should result. The graft should be sutured in place and firm pressure applied. The resistance offered by the skull is a valuable help to the pressure dressing in getting firm contact between raw surfaces. "Tulle Gras," as

recommended by Morax and others, is suitable to lay over the graft before putting on the gauze dressing and pressure bandage. A few layers of vaselin gauze will answer equally well as a covering for the graft.

RESTORATION OF THE CILIA LINE

There are several types of cases calling for restoration of the eyelash line. I should like to give four suggestive ideas:

1. In cases such as were mentioned under the head of severe ectropion, usually extensive loss of eyelashes results. Occasionally in



Fig. 5.—Loss of eyelash line. Eyelids held together by adhesions. Eye-ball intact. Bed prepared for graft at margin of upper lid. Hairy graft from brow about to be turned and placed at lid margin.

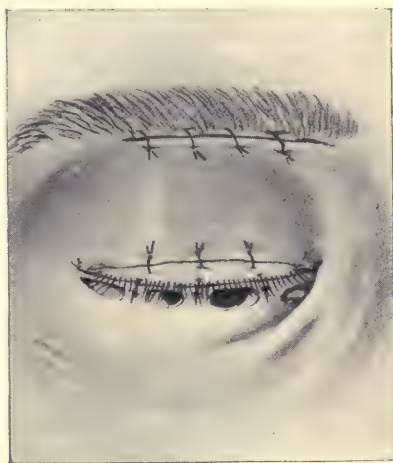


Fig. 6.—Hairy graft from brow sutured in position at lid margin. Brow wound closed by sutures.

extreme ectropion the upper lid margin is found in the brow, and it is possible to make the releasing incision in the lower part of the brow instead of below it, and so carry down a hair line at the time of the dissection made in preparation for the epidermic graft. This idea is decidedly worth bearing in mind.

2. Let us suppose a case of severe ectropion in which the lid margin is not actually in the brow and so the scheme just mentioned is not feasible, and let us suppose epidermic grafts have covered both upper and lower lids, and the lids are held together by adhesions in front of

the eyeball. Figs. 5 and 6 indicate the scheme to be recommended. The hairs should be taken as largely as possible from the nasal end of the inferior border, as here they are more numerous than at the temporal end and stand out in better direction. The idea in turning the graft upside down is to bring the hair line as near the lid border as

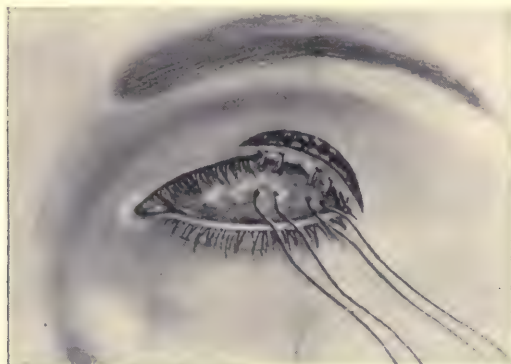


Fig. 7.—Loss of part of upper lid margin and eyelashes. Eyeball has been removed. Sutures introduced to unroll skin at injured lid margin and attach it to orbital contents.



Fig. 8.—Graft shown both before complete detachment from brow and after suturing in position at lid margin.

possible. A few weeks after the interpalpebral adhesions have been cut the lid margin should be trimmed off evenly close to the new cilia line.

3. In another type the eye may have been lost and a part of the lid margin with the cilia may have been destroyed. In such a case a

curved incision may be made near the injured margin and the skin below it dissected up. Then this skin is rolled back and tied down to the fundus of the socket with silk sutures, as shown in Fig. 7. Then a graft from the nasal end of the lower margin of the brow is placed so that the hairs will line up with the remaining cilia as accurately as possible (Fig. 8). It will be seen that the hairs of the graft really overlie the unrolled skin and so fall in line with the cilia. A little trimming of the margin of the lid may have to be done later.

4. Another possibility should be borne in mind. When skin of the upper eyelid is used for an ectropion of a fellow upper lid or a lower lid, it is feasible to carry the upper graft incision into the lower part of the brow and so carry with the graft a line of hairs to be placed for cilia. The graft below the cilia line may be made as large as desired. If this idea is used, in order to bring the hair line to the lid margin, the graft is carried directly to the denuded area, without turning, for the lower lid; but for ectropion of the upper lid the detached graft has to be turned upside down.

It would be absurd for one to expect a perfect line of eyelashes to result from any attempt at substitution, but patients are delighted with the imperfect results which can be obtained by the schemes suggested.

NOTCH AT LID MARGIN

A condition which we have to deal with occasionally is a deformity associated with laceration of the tarsus, manifesting itself in angulation of the lid margin and in destruction and distortion of cilia at the site of injury. This may be associated with ectropion or other deformity, but should be recognized as an entity. In a paper entitled "War Injuries of the Eyelids,"¹ I referred to a method of handling this condition and called the little procedure the "Halving Operation." The illustration (Fig. 9) gives the appearance of the deformity and the scheme of correction.

Operation.—The little mass of scar formation around the laceration of the tarsus is excised, and the tarsal flaps are cut cleanly across to give accurate apposition. Assuming that the deformity is in the upper eyelid, care should be taken to excise fully as much tarsus at the upper margin as at the lower in each flap of the tarsus. This is important, and even a slight overcorrection of the notch can be obtained, at the surgeon's discretion, by diverging the tarsal incisions from the lid mar-

¹ Trans. Amer. Ophth. Soc., 1919.

gin, so as to make the tarsal flaps slightly shorter above than below. The skin and orbicularis are cut so as to make a flap on one side, and a rectangular area of tarsus is exposed on the other. If necessary, to prevent tension on the wound, especially if the eyeball has not been removed, a canthotomy should be performed, and the branch of the external canthal ligament should be severed to release the tarsus. The little tongue of skin is trimmed enough for adjustment and a small triangular piece of skin may be removed above to avoid puckering. The conjunctiva and tarsal flaps are first sutured. A mattress suture is carried through the flaps, and tied after passing through a small rubber plate cut from sterile tubing. Sutures are introduced to give good apposition of the skin flaps. The mattress suture should be

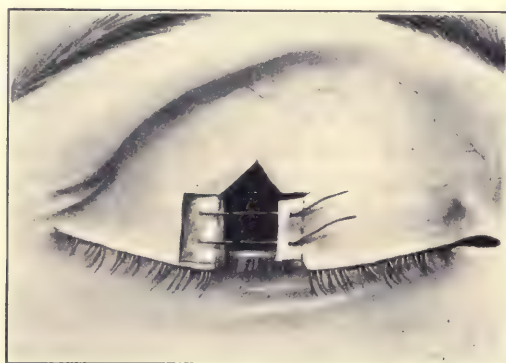


Fig. 9.—Scheme for correction of notch of eyelid.

removed in two or three days, and the others about five days after operation.

An important point is that tarsal wound and skin wounds should never be in the same position, but should be placed in such a way that there is overlapping. Thus, what is known in carpentry as halving is accomplished, and union is assured, even if there is a little sloughing of the skin. Furthermore, recurrence of the notch formation is prevented. After healing there is no break in the continuity of the lid margin or in the row of eyelashes. In handling notch cases different short-cut procedures have been tried, but the "halving" method has been the only satisfactory one in my hands.

In notch cases sometimes it is necessary to dissect out bands of scar tissue which lead from the tarsal laceration to the margin of the orbit.

There should be complete release, so that the lid margins will fall easily and completely together.

TRAUMATIC COLOBOMA OF LOWER EYELID NEAR INNER CANTHUS

A rather common injury is that of laceration of the lower eyelid through the canaliculus, extending downward and outward more or less accurately along the lower orbital margin. Improper primary healing of such a wound results in a coloboma near the inner canthus and ectropion of the lid near the coloboma. Complete correction of this deformity can be obtained by a simple manœuvre. Fig. 10 is

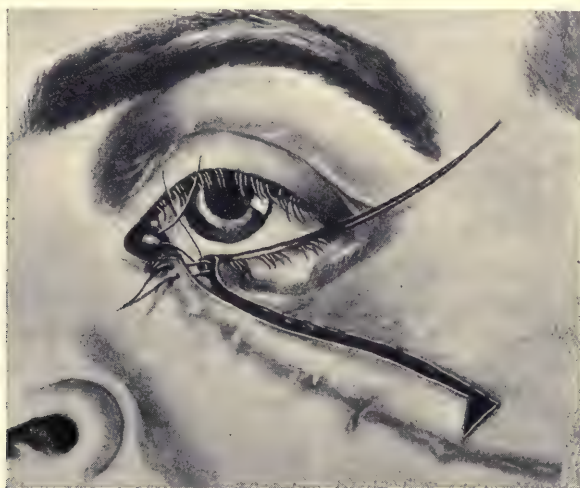


Fig. 10.—Operation for correction of coloboma of lower lid near inner canthus. To show sutures ready to tie at apex of flap.

from an unpublished series of drawings and is an attempt to show the way the dissection is made and the way the sutures are placed to make the correction. Scar tissue at and near the coloboma is dissected away. An incision is carried from the coloboma downward and outward. The attachment of the lower lid to the external canthal ligament is severed, and if necessary, another incision is carried outward and slightly upward from the outer canthus. The flap is undermined as much as necessary so that its apex can easily be carried to the inner canthus. The amount of dissection necessary will depend upon the amount of tissue loss at the coloboma. Sutures are placed

diagonally along the incisions in such a way as to advance the whole flap and have as little pull as possible for the sutures at the apex of the flap during the healing process. Of greatest importance is the adjustment of the tissues at the apex of the flap. The small amount of skin just external to the inner canthus is carefully and completely undermined. The skin and orbicularis are stripped from the anterior surface of the tarsus so as to expose 4 or 5 mm. of the tarsus. A suture is carried through the exposed tarsus, and then it is carried well into the internal lateral ligament or into the deep fascial tissue behind it so as to get a firm hold. A mattress suture mounted with two needles is carried through the exposed tarsus, then through the flap of skin which has been dissected up near the inner canthus (Fig. 10). When the sutures are snugly tied the apposition will be secure and the lid will lie well back against the globe and well up to the proper level, or even high enough to be in a position of overcorrection. Without this careful adjustment and definite overlapping of raw surfaces one cannot be sure of complete obliteration of the deformity.

RESTORATION OF OBLITERATED SOCKET

Many suggestions have been made for making new sockets after obliteration, and many disappointments have resulted from various sorts of procedures. Without entering into a discussion of the relative merits of the different operations designed for socket restoration, let me speak briefly of the method which I have adopted, and which I can recommend with confidence.

Epidermis has been chosen as the most satisfactory tissue for lining the cavity, as it will give a thin, pliable lining, free from hairs and an excess of oily secretion. The lateral aspect of the thigh is usually the most convenient place from which to take it. No elaborate preparation is necessary. The part from which the epidermis is to be taken is shaved and cleansed with alcohol and ether. Then with a long-bladed knife or razor with a keen edge, a large piece of epidermis is taken. For taking the graft I am now using the Stille (Norwegian) graft knife, and an interested barber gives it an exquisitely sharp edge, without which it is impossible to get a large graft free from perforations and free from true skin. This knife has a long blade, and is like a large table knife. The method adopted has been previously described in detail.¹

If the lid margins are adherent, they are separated by an incision.

¹ Amer. Jour. Ophth., July, 1921.

The dissection is then carried out in such a way as to separate the lids from the orbital contents. The following points should be borne in mind:

1. *The dissection must be kept superficial* so that in front of the dissecting knife or scissors there is only lid tissue, *i. e.*, only skin, orbicularis, the thin fascia of the lid, and the tarsus. It is not necessary to save the tarso-orbital fascia with the lid. Carrying the dissection back into the orbital tissue is probably one of the most common causes of failure.

2. Not only the superficial plane of the dissection, but *the extent and limitation of the dissection* are of importance. Temporally and below, the dissection should be carried well to the orbital margin or even 1 mm. or 2 mm. beyond it, as the graft must adhere to the periosteum of the anterior aspect of the orbital margin. On the nasal side the dissection should be extended to the anterior crest of the lacrimal groove and to the orbital margin above it. In dissecting at the inner canthus the caruncle should be saved if it has not been destroyed. The graft will adhere to the posterior surface of the caruncle and give it a permanent lining. In the division of the tissues above, the dissection should be carried behind the orbital rim but not necessarily to the roof of the orbit. In some cases the levator palpebræ superioris can be saved.

3. In preparing the bed for the graft *all cicatricial and granulation tissue should be removed*. Excision of granulation tissue is especially important as the contraction of such tissue may result in contraction of the socket. Reduction in size of the newly made socket is due to contraction of the underlying tissue rather than to contraction of the skin itself.

4. A common fault which manifests itself after restoration of the socket is that of too great *thickness of the lid margin*. This deformity can be obviated by cutting away tarsus. If the skin graft must extend completely to the margin of the eyelid, the tarsus may be split and thinned. If the graft is to extend nearly to the margin, sufficient tarsal plate may be cut away so that the graft will set in as an inlay. In any case, enough tarsus should be left to support the cilia, but not necessarily any more. A form should then be molded to fit the cavity. Dental impression compound is ideal for this purpose. Ordinarily for full restoration of the socket, the dimensions are approximately as follows: length 40 mm. to 45 mm., width 30 mm., thickness 5 mm.

The ideal graft for socket restoration is one without perforations

which is made up of epidermis only, free from layers of true skin, and large enough to be wrapped around the form of impression compound with generous overlapping. This means a graft $3\frac{1}{2}$ to 4 inches long and about 3 inches wide.

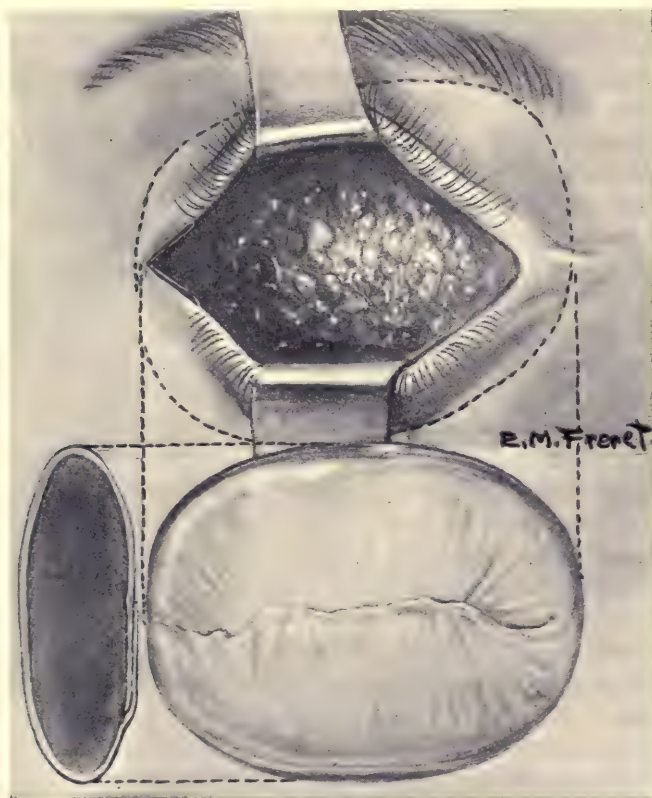


Fig. 11.—Restoration of obliterated socket. Eyelids retracted to show dissected cavity for socket. Orbital margins indicated by curved dotted line above. Anterior view and cross-section of form of dental impression compound covered with a single epidermic graft for lining of new socket.

The graft of epidermis is immediately wrapped about the form of impression compound, raw surface outward, and overlapped on the surface which is to be anterior. The form, completely covered with epidermis, is forced into the socket cavity (Fig. 11). It is not necessary to remove small blood-clots before placing it as they will not

prevent a take. The overlapping portion of the graft is placed forward so that if the edges are disturbed by manipulation they can be carefully replaced through the palpebral fissure so that every part of the form will be covered.

No sutures are used. A pressure bandage is applied, and over this adhesive strips. Very firm pressure is of importance to secure accurate contact at all points and to keep the cavity absolutely obliterated. This first dressing is left in place for a week. The form is left in place and is not touched for about three weeks. It is then removed and left out permanently. The artificial eye may be introduced at any time.

If the surgeon has been successful in carrying out the technic, the result will be a permanent socket, extending well beyond the canthi and of sufficient dimensions all around. The lids will be normally thin and pliable, and the thin-walled socket will not prohibit motility of the stump and artificial eye although the movements of the eye will be somewhat less than those following ordinary enucleation.

An interesting development is that usually lacrimal secretion will find its way into the new socket and maintain a comfortable moisture. As far as I know, trouble never results from activity of the lacrimal gland.

DISCUSSION

DR. W. E. LAMBERT (New York City): It has been my privilege to witness many of the operations which Dr. Wheeler has performed, and to follow up the end results, and I simply wish to express my admiration for the skill which he has displayed. He is rather modest in saying that the restoration of the lid margin adheres not particularly well. I recall one case which showed me that it was wonderful. Not only does it require a great deal of skill and painstaking, but I think one who does this kind of surgery must have a certain temperament.

DR. JOHN E. WEEKS (New York City): In former years, before asepsis was thoroughly understood, the great question involved in all of these plastic operations was how best to get the flap to take—what sort of a flap would be most liable to give the results desired. Reverdin, as we know, first taught the use of flaps without a pedicle by using minute particles of skin. Then Wolf followed with large skin flaps, and when these flaps were put in position without infection they usually took well, flaps with pedicles being safer than those without pedicles. The recent opportunity for great experience in plastic operations about the face, with our knowledge of asepsis, has served to revolutionize our ideas regarding plastic surgery about the eye and face, and our idea that flaps with pedicles must be used has changed entirely; our idea that no skin must be used has changed. With a knowledge of what has preceded the modern ophthalmologist bases his operation on the evidence that has been accumulated. Dr. Wheeler has not been hampered by previous

knowledge. He has overstepped the boundaries laid by men who have preceded him, and has produced results far superior to those previously obtained.

The principle of operating soon after the injury has occurred is one that he has changed entirely. Previously we had the impression that it was better to wait until the cicatrix was fully formed before we attempted to do plastic surgery. Dr. Wheeler has demonstrated that we may operate early and get most excellent results.

DR. J. M. WHEELER (closing): I have been asked many times how a large epidermic graft such as I have described can be taken. It requires some training and some patience to get the sort of graft that is necessary to cover a large area. I have found that the instrument makers will not give a keen enough edge to a razor or knife to take the proper kind of graft. The edge must be extremely keen and evenly keen. If there is any place that is not sharp, you are sure to have trouble. An interested barber can get a keen edge on a grafting knife if you explain to him what you want, and he will take special care because he will feel that it is the most important work he has ever done.

LAGOPHTALMIE BILATÉRALE CONSÉCUTIVE A LA PERTE ACCIDENTELLE DE LA PEAU DU FRONT, ET DU CUIR CHEVELU. BLÉPHAROPLASTIE

DOCTEUR J. N. ROY

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Montreal, Canada

OBSERVATION

Le 28 octobre 1920, la soeur M. J. âgée de 37 ans, vient nous consulter pour une lagophtalmie double, remontant à près d'un quart de siècle. Elle raconte qu'à 12 ans, un jour qu'elle travaillait dans une buanderie mécanique, sa chevelure fut prise dans une courroie qui la souleva jusqu'au plafond. Sa tête étant venue en contact avec un soliveau, elle fut instantanément scalpée, et retomba sur le plancher, sans avoir perdu connaissance, et sans avoir à déplorer de fractures. La plaie frontale se limitait aux sourcils qui étaient arrachés, celle de la région temporale faisait une encoche à la partie supérieure du pavillon des oreilles, surtout à droite, et à part un peu de cheveux qui restaient à la nuque, toute la peau du crâne était disparue. Les paupières supérieures, n'étant plus attirées en haut, se retournèrent et recouvrirent les fentes palpébrales. La douleur fut naturellement très vive, et l'hémorragie considérable. Des linges humides froids

furent immédiatement appliqués en attendant le médecin. A son arrivée, celui-ci fit un pansement antiseptique à l'acide phénique. Durant les quatre semaines qui suivirent l'accident, des hémorragies assez fréquentes se produisirent. Le périoste était partout intact, et les pansements humides à l'acide phénique furent continués pendant trois mois. Après cette période, ils ne furent faits que la nuit, et le jour on appliquait de l'acide borique en poudre, tout en laissant le crâne à découvert. Peu de temps après ce nouveau genre de pansement alternatif, un érysipèle se déclara, et faillit emporter la malade. Environ six mois après le traumatisme, la plaie commença à s'améliorer et les paupières, grâce aux pansements compressifs appropriés, se replacèrent petit à petit; toutefois les cantus externes eurent dès le début une tendance à être attirés en haut. Cette traction s'augmentant toujours par l'épidermisation de la peau du front et de la tempe, fit qu'à la fin de la première année, la malade ne pouvait plus fermer les yeux. Huit ans après l'accident, une kératite double par lagophtalmie se déclara, surtout localisée à l'oeil gauche. L'oculiste consulta le traitement ordinaire, sans oublier naturellement le bandeau que la malade ne cessa jamais de porter la nuit depuis cette époque, et la guérison fut complète au bout de deux mois. La région fronto-temporale fut très lente à guérir, puisqu'elle prit cinq ans à se cicatriser. Pendant cette période, la lagophtalmie augmenta toujours, et les paupières supérieures devinrent presque immobiles. Les oreilles furent attirées en haut. La région occipitale s'épidermisa assez rapidement. Quant au vertex, il fut particulièrement rebelle à la guérison, bien que les pansements humides à l'acide phénique fussent repris et toujours continués depuis son infection érysipélateuse. Dix-huit ans après l'accident, un eczéma envahit la région fronto-temporale, et prit une année avant de disparaître. Comme à ce moment la plaie du crâne semblait vouloir s'éterniser, un chirurgien pratiqua la greffe de Thiersch. Il fit aussi une incision dans la région du cantus externe de l'oeil gauche, des tractions et des pansements appropriés, dans le but d'abaisser la paupière supérieure correspondante. Malheureusement ces deux interventions ne donnèrent aucun résultat, car une seconde infection se déclara, et les greffes furent éliminées. Un mois après cette première tentative, le chirurgien essaya sur le vertex la greffe dermo-épidermique en flots, qui cette fois fut tolérée. Pendant les six années qui suivirent, l'amélioration progressa très lentement, sans toutefois donner un résultat complet. A plusieurs endroits, la cicatrisation ne voulait pas se faire,

des croûtes se formaient pour tomber ensuite, et malgré les pansements humides, il y avait toujours de la suppuration.

A l'examen des yeux, nous constatons deux petites taies des cornées. La vision est cependant excellente après correction d'un léger astigmatisme hypermétropique.

Quant aux paupières, il existe une lagophthalmie mécanique des plus considérables. Les cantus externes, décollés de la conjonctive oculaire, sont très fortement attirés en haut, et la traction est encore

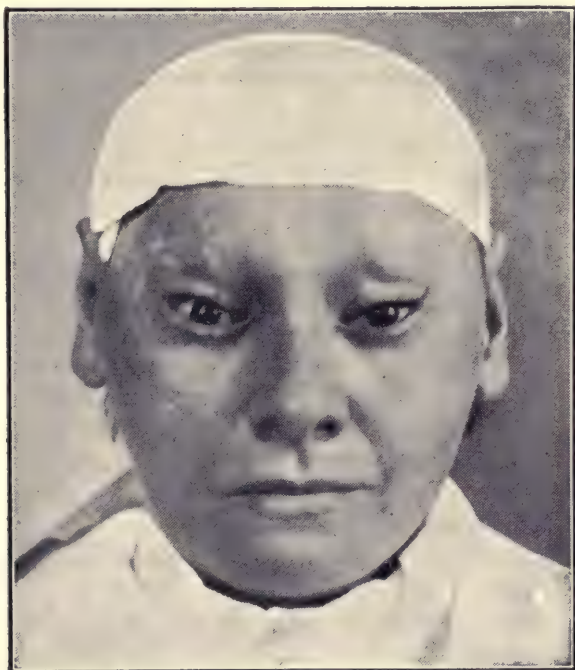


Fig. 1

plus prononcée à droite qu'à gauche. L'aspect de la malade rappelle, d'une manière exagérée, le type caractéristique de la race mongolique. Lorsqu'elle fait un effort pour fermer les yeux, les paupières bougent à peine. Il n'y a pas d'ectropion.

Les sourcils sont entièrement disparus.

La peau du front et des tempes est très mince, sans tissu adipeux sous-jacent, et parcourue par un grand nombre de petits vaisseaux sanguins superficiels.

L'oreille gauche est remontée de quinze millimètres environ, et la droite de deux centimètres.

Il y a sur le vertex, encore incomplètement cicatrisé, une dizaine de petits endroits remplis de bourgeons, ou recouverts de croûtes.

La région occipitale est parfaitement guérie, et il existe huit centimètres de cuir chevelu à la partie inférieure de la nuque.

Après nous avoir fait part des ennuis résultant de la lagophthalmie, la malade nous explique jusqu'à quel point est désagréable la sensation de traction continuelle de ses cantus externes. Aussi est-ce avec une certaine anxiété qu'elle nous demande si nous pouvons la guérir. Sur notre réponse affirmative, et après l'avoir mise au courant de ce qui devait être fait dans son cas, elle accepte immédiatement l'opération qui, pour des raisons personnelles, est fixée au 4 décembre. A cette époque, l'apparence de la patiente est celle indiquée par la photographie No. 1.

OPERATION

Après avoir décidé de commencer par le côté gauche, toute la partie sur laquelle nous devons intervenir étant parfaitement stérilisée, nous faisons à la région fronto-temporale et sur la joue, une série d'injections de novocaïne-adrénaline. Une incision de cinq centimètres environ est ensuite pratiquée, se terminant un peu en bas et à onze millimètres du cantus externe. Cette incision est très légèrement concavo-convexe, à direction inférieure, et se trouve à traverser l'emplacement du tiers externe du sourcil arraché. Après avoir largement disséqué toute la région environnant le cantus, nous réussissons à abaisser suffisamment les paupières pour leur donner une position tout à fait horizontale. Afin de combler cette plaie entr'ouverte, nous prélevons sur la joue un lambeau de sept centimètres environ de longueur sur quinze millimètres de largeur dans son plus grand diamètre qui, une fois détaché, est mis en place. Dans le but d'immobiliser le cantus, et d'empêcher qu'il remonte de nouveau pendant la cicatrisation post-opératoire, nous l'attirons en bas au moyen d'un catgut passé dans le périoste de la région malaire. Le lambeau fronto-temporal est soigneusement suture à la soie, et après avoir décollé la peau adjacente à l'incision jugale, les lèvres de la plaie sont facilement coaptées et maintenues en place au moyen de crins de florence. Finalement nous appliquons un pansement légèrement serré, et le plaçons de telle sorte que la compression se fasse de haut en bas. Les suites opératoires sont des plus simples, et la plaie guérit par première intention.

Comme la malade est très désireuse d'être rétablie le plus tôt possible, nous intervenons à droite dix jours après la première opération. La technique suivie étant tout à fait semblable pour les deux côtés, nous ne croyons pas devoir la décrire de nouveau. Cependant comme le cantus externe était un peu plus relevé à droite qu'à gauche, sa dissection fut plus laborieuse pour parvenir à un abaissement complet. Encore cette fois, les suites opératoires évoluent sans incident et nous obtenons une réunion par première intention. Un soin par-



Fig. 2

ticulier est apporté aux pansements, et grâce à la plus ou moins forte compression pratiquée de haut en bas, nous augmentons l'effet de notre opération, et réussissons à avoir deux fentes palpébrales parfaitement symétriques et horizontales. La malade peut maintenant fermer les yeux sans effort, et comme le releveur des paupières supérieures n'avait pas été lésé lors de l'accident, elle peut aussi les ouvrir normalement. La conjonctive palpébrale des cantus externes est en contact avec la conjonctive oculaire, et il n'existe aucune trac-

tion disgracieuse de cette partie. A la fin de décembre la patiente est assez bien pour retourner dans sa communauté.

Naturellement nous avons profité de son séjour à l'hôpital pour traiter ses petites plaies craniennes au moyen de pansements humides, de curettages des bourgeons et d'applications de teinture d'iode. Ce traitement, continué dans la suite, amena une forte amélioration



Fig. 3

sans toutefois donner une guérison complète. Pour atteindre ce but, il faudrait, croyons-nous, pratiquer encore quelques greffes dermo-épidermiques, vu la très mauvaise nutrition de tout le tissu cicatriciel crânien.

Le 26 février et le 8 mars 1921, nous faisons sous anesthésie locale, une petite autoplastie des pédicules de nos lambeaux indiens. Comme ceux-ci avaient été taillés avec assez de précision, et qu'il n'existait pas de bourrelets cutanés à la région sourcilière, nous ne sommes pas obligés d'y faire de retouches.

Après guérison complète de ces deux plaies, nous proposons à la malade de tenter la greffe des sourcils que nous aurions prélevée sur ce qui restait de cuir chevelu à la partie inférieure de la nuque. Etant donné que son lambeau frontal, porté dans sa communauté, en masque l'absence, et qu'elle est enchantée du résultat opératoire obtenu, elle refuse cette dernière intervention. Les paupières, sur un plan bien horizontal, se ferment avec la plus grande facilité, et les yeux s'ouvrent sans effort—, d'ailleurs comme il peut être constaté sur les photographies 2 et 3, prises le 7 avril.

L'opérée, revue en décembre 1921, est toujours dans le même état, et à peine existe-t-il quelques traces de ses incisions cutanées.

En publiant cette observation, nous n'avons pas la prétention de préconiser une méthode nouvelle de blépharoplastie. Nous nous sommes simplement inspiré de l'expérience personnellement acquise pendant la récente guerre, en fait de chirurgie de la face et des paupières, en choisissant l'unique procédé rationnel dans le cas présent. En effet, avec la greffe dermo-épidermique, nous aurions probablement couru vers un échec, car, même si elle avait été pratiquée d'une manière impeccable au point de vue de l'asepsie et de la taille d'un lambeau suffisamment grand, il ne faut pas oublier que notre intervention portait sur un tissu très mince et mal nourri.

Afin de sauvegarder l'esthétique de la face, nous aurions pu nous servir du procédé de Snyder de Chicago, en prenant un lambeau pédiculé sur le cou. Toutefois ce procédé aurait produit un traumatisme opératoire beaucoup plus considérable que celui employé chez notre patiente, et n'aurait pas eu sa raison d'être dans le seul but d'éviter une cicatrice jugale maintenant à peine perceptible.

Comme il était nullement question dans notre cas de recourir à la méthode italienne, il nous restait donc, pour combler l'espace produit par l'incision et l'abaissement du cantus externe, qu'à prendre sur la joue, au voisinage immédiat de la plaie, un lambeau approprié, ce qui revenait à choisir la méthode indienne. D'ailleurs les différents procédés de blépharoplastie sont maintenant bien connus, surtout depuis les remarquables travaux de Morax de Paris, qui est certainement l'oculiste possédant la plus grande expérience en chirurgie palpébrale. Nous-mêmes avons eu l'occasion de publier un certain nombre de mémoires sur les greffes osseuses et cartilagineuses,—que dans la présente communication, nous passerons sous silence—et sur les autoplasties de la face en général, dans lesquels nous avons exposé les meilleures méthodes à préférer dans chaque cas particulier.

Pour celui-ci, nous attirons l'attention sur le fait de nous être servi de deux catguts passés dans les cantus et attachés au périoste malaire, ce qui a grandement contribué à les stabiliser. Le pansement, comprimé de haut en bas, a également augmenté l'effet de notre intervention.

Si la malade en avait manifesté le désir, nous aurions volontiers essayé de lui greffer des sourcils, sans toutefois être certain du succès, étant donné la mauvaise qualité de la peau et des tissus sous-jacents, qui auraient eu le rôle de nourrir ces greffes. Aussi, en présence du résultat obtenu, nous avons lieu d'être satisfait du procédé choisi.

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LIGHT-SENSE: THE PRACTICAL SIGNIFICANCE OF ITS VARIATIONS: SIMPLE TESTS FOR DETERMINING THEM

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I deeply regret that I have been unable to attend this International Congress and that I have missed this opportunity of learning so much from ophthalmologists of world-wide reputation. I would now express my thanks to the Committee for allowing me to present this paper for your criticism in my absence.

Light-sense is the faculty of recognizing different luminous intensities. Too little attention has been paid to the sensibility of the eye to variations in the light stimulus, although this may be of extreme importance in certain services. We are asked for a report on a candidate's sight, and we send a report of his visual acuity; *i. e.*, of his macular form-sense (without and with correcting glasses, should he require any). A man may have a visual acuity of 6/6 and yet unless we examine his light-sense we may miss the fact that he is night-blind. Of what use is such a man, when on watch at sea, in detecting an object, whether a derelict or an iceberg, in the ship's course at night?

You will all have noticed at nightfall, when there is no moon, that as the light begins to fail that red colors become much darker, and presently a red geranium will appear quite black and the grass grayish, although yellow and blue flowers can still be seen; for a time the blue flowers appear extraordinarily brilliant, as all other colors are unrecognizable, and then finally, as the darkness deepens, blue disappears, and one becomes totally color blind. This successive disappearance of colors is called Purkinje's phenomenon.

When this stage of dark-adaptation, or *scotopia* as it is called, is reached, one can still dimly see objects by their differing depths of grayness, but it will be found that one sees best by looking a little to one side of them. Arago first called the attention of astronomers to

this point, telling them that they could best get a glimpse of a very faint star by looking a few degrees away from it. It will be found that in complete scotopia the fovea is a blind spot; *i. e.*, there is a central scotoma in the rod-free area of the retina.

Normally with full illumination the visual acuity or form-sense diminishes very rapidly from the fovea toward the periphery. With a foveal acuity of 6/6, at 5° distance the acuity is 6/24, at 10° 4/60 and so on. In a very dim illumination light sense is absent at the fovea, and is at a maximum about 10° from the fovea.

	Fovea	5°	10°	15°	
Form-sense.....	6/6	6/24	4/60	2/60	Full illumination
Light-sense.....	0	Good	Max.	Good	Dim illumination

Now the fovea subtends an angle of about 1° and the rod-free area an angle of about 3° at the second nodal point of the eye; beyond this area rods appear in increasing numbers and cones become less and less as one proceeds towards the periphery. One is, therefore, led to think that rods, or something associated with rods, must be the percipient elements for light-sense.

Comparative anatomy supports this view. With one exception all nocturnal animals possess an excessive number of rods which are especially long, and a very ill-developed fovea, whereas all diurnal animals have a well-developed fovea and a far greater number of cones. A very striking example of this peculiarity is found in the family of Saurians. All the diurnal lizards (*e. g.*, the chameleon) possess only cones, while the nocturnal lizards or geckos have only rods.

The exception mentioned above is the nocturnal tortoise which has only cones; but the tortoise is peculiar, it moves so slowly that it can neither escape from its enemy nor pursue its prey, and it is quite possible that it may be guided to the insects and plants it eats by scent alone.

When we pass from bright sunlight to a very dimly lighted room we can see nothing until our eyes have become scotopic or adapted to the dark. In order to obtain full dark-adaptation it is necessary to remain in an absolutely dark room for three-quarters of an hour or so, and then some very curious phenomena are found. In scotopia the light-sense is about twice as great with both eyes as with only one, again if

an object be illuminated by a dim composite white light the retinal sensibility is proportional to the square root of the area stimulated. In light-adapted eyes there is no such binocular summation of stimuli, nor, if the retinal area stimulated exceeds $1'$, is there any increase of brightness from stimulating a larger area.

Prof. E. M. Barnard some years ago caused great consternation among the pundits of ophthalmology owing to their ignorance of this fact. He found that he could detect a wire at such a distance that its diameter only subtended an angle of $0'.44''$. Most people jumped to the conclusion that his minimum visual angle was less than half a second and that it was his form-sense that was being tested. The minimum visual angle for form-sense is $53''$, and for convenience is usually assumed to be $1'$, so many discredited his observation. This was absurd for Prof. Barnard is known throughout the world for his reliable and accurate observations at the Yerkes Observatory. We can see that it has nothing to do with his form-sense, it was only a proof of his great sensibility to light difference. If you try, as I have done, putting up a fine wire 16 feet long horizontally against the sky, you will see it quite easily at a considerable distance, but if you block out from your view all but a foot of it, you will not see it at all, as its effect on your retinal sensibility is only one-fourth of what it was, thus showing that as it obeys the square root area law it must be a light-sense, not a form-sense, test.

The subject is a very difficult one, as most of our so-called form-sense tests are really composite tests of our light-sense also; as, for instance, especially the common dot tests and even Landolt's excellent broken ring tests.

Now visual purple or rhodopsin occurs according to Kühne in the rods only, and there is good evidence to show that rhodopsin plays a very important part in the physiology of light-sense. When a spectrum of very low intensity is viewed in the dark by a scotopic eye, it appears as a colorless gray streak, the brightest part of which is in the green ($\lambda = 5300$ A. U.) and not about $\lambda = 5800$ A. U. in the neighborhood of the D line, as is the case in the normal photopic eye. The luminosity curve seems to be bodily shifted toward the violet end of the spectrum as the red end is shortened, it is in fact exactly similar to the luminosity curves of the totally color blind. Then again the bleaching effect of light on a frog's rhodopsin is greatest when it is of wave length 5300 A. U. (in the green). For these and further details I would refer every earnest inquirer to Parsons' excellent "Color

Vision" (published at the University Press, Cambridge), to which much of this paper is due.

You will, however, be wearied with all this academic stuff, and be anxious to know what practical bearing it has on diagnosis, so I turn now to this aspect of the subject.

Light-sense is tested in two different ways: (1) The discovery of the smallest quantity of light that can be recognized on a black background; this is called the *light minimum* test, and is always referred to as L. M.; and (2) the smallest difference of illumination that can be appreciated between two sources of nearly equal luminosity. This is called the *light difference* test and is always denoted by L. D.

Now the important practical point is that the sense for L. M. is always most affected if the *receptive* part of the retina is most implicated; *i. e.*, in affections of the bacillary layer, the visual purple, and the choroid on which the efficiency of the visual purple depends; whereas the sense for L. D. fails if the *conductive* part of the visual function is primarily at fault. For instance, in optic neuritis and in retrobulbar neuritis L. D. is at fault. An easy memoria technica is to associate L. D. with o. d., the optic disc, but I wish to emphasize the point that L. D. fails also when the nerve fibrils in the retina are chiefly affected. This I have only recently found out. In all previous publications that I have seen on the subject it has been stated that defective light difference was diagnostic of a lesion in the optic nerve. This hasty assumption was no doubt due to the very troublesome apparatus that was used for testing light-sense. With my simple little rotating disc the whole examination need not take longer than three minutes, and so more experience of the diagnostic value of light-sense phenomena can be gained in a week's hospital practice than could be formerly attained in a year, unless one's whole time were devoted to the subject.

I long ago found that toxic amblyopia was always associated with a L. D. defect, and yet we most of us suspect the lesion to be in the ganglionic layer of the retina. It was Dr. Traquair, to whom I pointed out my difficulty, who suggested the association of L. D. defects with lesions of the conducting apparatus instead of simply confining them to those of the optic nerve. I have verified this suggestion by finding L. D. defects in several cases of retinitis. I now think that if the superficial layers of the retina are affected a L. D. defect will be found, but if the deeper layer (of the rods and cones) or if the choroid be at fault there will be a L. M. defect.

In all the cases of early glaucoma that I have examined in this way, I have always found a L. M. defect as the earliest symptom, and that it was afterwards followed by a L. D. defect. I suggest as an explanation of this that the bacillary layer is more sensitive to slight pressure than the nerve-fibers. My observations need most careful confirmation, and I would be exceedingly grateful to any of you if you can confirm or refute them. It is most important to obtain a really reliable indication of the first onset of chronic glaucoma, and I believe that the light-sense test will be found to be far more trustworthy than any single tonometric reading. I think that too much reliance is now placed upon the tonometer; we all know that there are fairly wide physiologic variations from what is regarded as the standard reading. On physical grounds the reading must depend upon the stiffness of the cornea, on the size of the eyeball, etc., as well as upon the tension of its contents, so that although a series of tonometric readings taken every week on the same case has a very great value, undue weight should not be given to one isolated observation.

In cases of early cataract or hazy media when no details of the fundus can be seen with the ophthalmoscope, it is often of extreme importance to obtain some indication of the condition of the structures at the back of the eye. An examination of the light-sense of the patient helps us out of the difficulty. If you view these rotating discs through dark-tinted glasses, you will find that both your L. M. and your L. D. perception is as good as without the dark glasses. This is no doubt due to the partial scotopia induced by the dark glasses. If the obscuration is very great, the L. M. and the L. D. perception will fail almost equally.

Two months ago I was consulted by a new patient for cataract who told me that now she was quite sure that her right eye was ready for operation. With the ophthalmoscope I could only see a very dim red reflex, but on testing her light-sense I found that both L. M. and L. D. perceptions were very defective but especially her L. M. sense (L. M. less than 12.5, L. D. = 50, according to my nomenclature where the standard is L. M. = 200, L. D. = 100). I concluded that there was probably a macular hemorrhage present, as projection was good, or fairly good. Needless to say I did not recommend an operation on that eye, and it was the light-sense test that saved me from a disastrous operation.

We know that, unlike form-sense, light-sense is fairly good even to

the periphery of the field, and this peripheral or rod vision is of far more importance than most people think. All must have noticed how very readily one notices a movement of any object in the peripheral field, and so the eyes are turned in that direction to see clearly with the maculæ what it is. Indeed, as the projection from the periphery is so good it is often unnecessary to turn one's eyes toward the object. In a town one avoids bumping up against passers-by not by macular vision but by peripheral or rod vision, *i. e.*, by light-sense not by form-sense. Without good light-sense one will not see what to look at. The British Board of Trade ordains that seamen should pass a very high standard of tests for macular visual acuity, but pays little regard to light-sense. Quickness of sight—to see an iceberg in twilight, for instance—is what is wanted, not ability to determine details about its shape that subtend angles of one minute.

Light-sense is the most primitive of the visual functions and is, therefore, the last to be lost in ordinary cases of amblyopia. At any rate in children with an amblyopic squinting eye in which the form-sense may be diminished to 6/36 or even less, its light-sense will usually, if not always, be found very good, often better than in the normal eye; however, in later life this extraordinary light perception seems to be lost.

There are some curious points about night-blindness that I have not the time here to discuss, for these and other matters I would refer you to a paper of mine on Light-sense published in the Transactions of the Ophthalmological Society of the United Kingdom, vol. xl, 1920.

PERCIVAL'S LIGHT-SENSE TESTS

The test can be made in ordinary daylight by rotating these celluloid discs on any convenient rotor or by spinning them on an ordinary dissecting needle.

L. M. DISCS.—There are two black discs, on each of which are three white sectors of different sizes, when one of these discs is rotated three lighter rings are seen of which the inner is the lightest. On the disc with the large sectors, the inner ring denotes a L. M. of 1/12.5, the intermediate ring 1/25, the outer 1/50, which it is more convenient to denote by 12.5, 25 and 50 as ability to see the outer ring denotes a better light minimum sense than that which can only see the inner ring. Similarly the disc with the smaller sectors denotes L. M. of values 50, 100 and 200.

L. D. DISCS.—These are white discs with black sectors of correspondingly different sizes, and when rotated indicate the light difference sense for values of 12.5, 25, 50 and 50, 100 and 200.

It will be noticed that a light-sense of either kind, of value 50, is given twice; this is often useful in forming an opinion of the reliability of the statements of the patient.

It will be found that few can distinguish the faint gray ring that corresponds to L. D. = 200, and so I assume that the standard for L. D. is 100, while that for L. M. is 200.

It might be hastily assumed that the size of the black sector denoting L. D. = 100 should be of angular aperture $\frac{3.60}{1.00}^\circ$ or $3^\circ 36''$, but as Abney has shown us that 5 per cent. of the incident light is reflected from lamblack, we must add $1/19$ to the angle, so that this sector must be of $3^\circ 47' 22''$; similar additions have been given to the other sectors.

DISCUSSION

DR. C. E. FERREE (Bryn Mawr, Pa.): Mr. Percival expresses the hope that his conclusions with regard to the light difference and light minimum will receive a wider confirmation than he has given them. It may be of interest in this connection to note that we are just beginning a long study of the application of functional testing to the diagnosis of eye diseases at the Polyclinic Hospital of the Graduate Medical College of the University of Pennsylvania. This study will include, among others, determinations of the light minimum and light difference, also the color minimum and color difference, with apparatus which permits of finely graded changes of intensity and the exact specification of the amounts of light used in every case.

In Mr. Percival's test for the light minimum the task set for the patient is the discrimination of the gray just noticeably lighter than black (coefficient of reflection, 4–6 per cent.) with the eye adapted or sensitized to high or daylight intensities of illumination. The light difference is determined under the same conditions of adaptation. This can scarcely be said to test the eye for scotopic or twilight vision. Of course these two determinations can be made with the eye in any state of adaptation and it may be of great diagnostic value to make them with the eye in a photopic state of adaptation as is done in Mr. Percival's tests; however, since a disturbance of the process of dark-adaptation is believed by many to be characteristic of certain types of disease, it seems to me to be important to make the test also with the eye dark adapted. This does not seem to be possible with the discs described by Mr. Percival. Mr. Percival's tests also should be made with a constant intensity of light on the discs. The Fechner fraction or the ratio expressing the value of the light difference is independent of the intensity of illumination only over comparatively small ranges of change.

The type of disc used by Mr. Percival was first described by Masson in 1845. It was much used by the earlier psychophysicists: Fechner, Aubert,

Delboeuf, Helmholtz, Müller, Kraepelin, Schirmer, and others, for the determination of the light difference. Its advantages for this purpose in clinic work are its very great simplicity and the speed with which it can be used. Some of its disadvantages are its inflexibility of gradation and the fact that it cannot be used for dark adaptation,—or over wide ranges of illumination, without the provision of many gradations, or rings of gray representing the needed differences in brightness from the background. It is also fit only for the determination of the light difference. The light minimum cannot be determined by this type of device. The determination of the light minimum should be made in the dark room, and in the strict sense of the term requires a fully dark-adapted eye.

I would suggest as tests for the use of the British Board of Trade acuity at low illumination and speed of discrimination both of the acuity object and of the light difference at low illumination. We have devised and used apparatus for making these determinations and have found them to be of great value for selecting eyes as to fitness for vocations requiring speed and accuracy of seeing at low illuminations. Eyes having the same acuity at high illuminations will by no means always be rated as equal at the low illuminations and the scatter is very much greater still when speed is added to the requirement at low illumination. Speed and power to sustain are aspects which have been very much neglected in the testing of acuity and other ocular functions. When added to the test they not only greatly increase its sensitivity, but take account of functions which are of the greatest importance to the working eye. Errors in refraction, for example, which cannot be detected by the conventional method of testing acuity, show plainly in acuity at low illumination, and in speed and power to sustain at any illumination, more particularly at low illumination. While these extraordinary tests may not at this time be considered feasible for office and clinic work, the results of investigations in which they have been used should teach us not to be too well satisfied with our present standards of accomplishment in the detection and correction of defects.

DR. OTTO ROELOFS (Amsterdam, Holland): I was surprised at hearing that in dark-adaptation and binocular vision there is a summation of stimuli which would fail by light-adaptation. This conception, first defended by Piper, is not right. Several investigations, principally made at the University of Amsterdam, showed that this summation does not exist, neither by light-adaptation nor in dark-adaptation. The phenomena found by Piper are the results of a fault in his examination. If we take care that the adaptation of both eyes is perfectly equal it is impossible to find any summation of stimuli. For all that it is true that the threshold for light-perception (the light minimum) is lower by binocular vision than by monocular vision; this fact wants another explanation, which I cannot discuss now because it would take too much time. I only wish to mention that this difference between binocular and monocular vision can be found as well by light-adaptation as by dark-adaptation. These results are confirmed by the investigations of Lehmann.

I want to ask the attention to the connection between apparent disturbances in the sense of light and disturbances in the field of vision, and I should like to consider the possibility that a disturbance in the center of the field of vision will give in the first place a disturbance of the light difference, because there it is examined by more *light*-adaptation, and that a disturbance in the periphery of the field of vision will give a disturbance of the light minimum because it is examined by more *dark*-adaptation.

I must say that the method of examination does not satisfy me. We always must have the same dark- or light-adaptation as a starting point for our examination of the sense of light. If we are not sure that at the beginning of every examination the dark- or light-adaptation is perfectly the same, our results cannot be of any significance. Especially I am surprised that a cataract operation can be refused on the result of such an examination. This method of examination requires not only a certain degree of sense of light, but also a certain degree of visual acuity; we have to discern the several bands on the rotating disc. An opaque lens is not the same as a dark-tinted glass.

COL. R. H. ELLIOT (London, England): I want to express my dissatisfaction with the present state of our knowledge of light-sense, and of our tests for light-sense. I think we need much more spade-work before we can be satisfied. Both the central and peripheral fields require to be tested. We see cases of glaucoma obviously suffering from a deficient light-sense in the peripheral field, and yet having excellent central appreciation both of light minimum and of light difference. It is this divergence, or apparent divergence, of my experience from that of Mr. Percival and others that has led me to speak. I suggest that the appreciation of light by the glaucomatous eye follows the variations in the visual field, whether central, paracentral or peripheral, and that just as we may find a patient with good central vision, but with damage to his paracentral or peripheral visual fields or both, so his light-sense over the same areas may be affected. Chronic glaucoma may be expected to strike first at the paracentral area, and next at the periphery of the field. Congestive glaucoma will attack the whole field from center to periphery in light-sense, just as it does in the other functions of the eye.

My preference is for a light-sense apparatus working with an actual light, and Zeiss has made one such for me, which I think will prove satisfactory for a clinical test both of light minimum and light difference.

Are we safe in neglecting all dark-adaptation of our patient in advance? It seems doubtful, and I think we need expert guidance on this subject.

DR. ALEXANDER DUANE (New York City): Mr. Percival in his paper reviews some elementary principles in the physiology of the light-sense, calling attention especially to the following facts:

1. The rods are particularly associated with this function.

2. Hence, the rod-free portion of the retina, *i. e.*, the fovea, is undersensitive to differences in illumination, and therefore when the illumination is considerably reduced ceases to function, so that a central scotoma develops.

3. In the peripheral portions of the retina, on the other hand, in which the form-sense is so weak, the light-sense is strong. It reaches its maximum in a zone some 10 degrees out from the fovea, but is good on either side of this zone. It is hence especially important in determining the capacity for peripheral vision.

4. The light-sense is the most primitive of the retinal functions and is hence found well developed in conditions in which the form-sense is very deficient; for example, in strabismic amblyopia. Moreover, in normal subjects it is well retained even when the illumination is so considerably reduced that the form-sense is notably impaired.

5. Some of our so-called form-sense tests, in other words, tests of visual acuity, are really in part tests of the light-sense. This applies to the ordinary letter tests. Failure to recognize this fact has led to some erroneous conclusions. Thus because Prof. Barnard could recognize a wire against the sky at a distance which made the wire subtend an angle of 0.44 second, it was argued that this represented his minimum visual angle, which would mean that his visual acuity was 125 times that usually regarded as normal. As a matter of fact, however, the test was a test of light-sense only and no more represented a test of visual acuity than is the ability to distinguish a fixed star which, practically speaking, subtends no angle at all.

Turning from these theoretical considerations to practical points, we may make the following statements:

1. The tests of the light-sense are important clinically in two regards: (a) The light-sense being concerned especially with peripheral vision and with the discrimination of objects affording but a faint contrast with their surroundings it is important that it should be systematically examined as a routine matter in those who in their avocations have to discriminate objects situated in the periphery of the field and but faintly visible. It is important, that is, to test it in sailors, particularly in lookouts and signalmen, railroad men, chauffeurs, aviators, etc. Of its great significance for the Army and Navy service I can personally testify. (b) The test is important in determining the presence of diseases of the sensory apparatus of the eye and in discriminating between different affections of this apparatus. In this regard we must note that mere opacities of the media, unless very dense, do not greatly diminish the light-sense.

2. In making tests of the light-sense we must distinguish between the light minimum (LM) and the light difference (LD). The former is measured by the smallest amount of light that can be distinguished in contrast with darkness; the latter by the smallest difference that can be appreciated between two contrasting light stimuli.

3. The light difference is particularly affected in diseases of the conducting apparatus, that is, the optic nerve and, according to Percival, the prolongations of the latter in the retina. Lowered light difference, therefore, is found in optic neuritis and atrophy, retrobulbar neuritis, and toxic amblyopia; probably also generally in affections involving specifically the superficial layers of the retina. The light minimum, on the other hand, is lessened in choroidal affections and in the deeper forms of retinal disease. Percival finds it affected quite early in glaucoma, the light difference not failing until later.

4. In affections of the media so dense as to affect the light-sense, both light difference and light minimum fail and fail equally. Here a marked disproportion between the two in the case, for example, of a cataract, indicates a disease either of the light-perceiving or light-conducting apparatus—retina or optic nerve. Percival cites an interesting case of cataract in which he declined to operate because the presence of such a disproportion made him infer, correctly, the existence of a macular hemorrhage.

5. The appliances that he uses for testing the two are exceedingly simple. They have the great advantage that they do not require a long preliminary period of dark-adaptation, and hence can be used by daylight. Moreover, the examination can be completed in one or two minutes.

6. The tests themselves are simply the well-known rotating discs, of which the Maxwell disc is a type. Their special significance consists in the way they are graduated so as to indicate the normal, or definite fractional proportions of the normal. The test for the light minimum consists of a black disc which has on each side three white sectors. The innermost sector on the back surface occupies $1/12.5$, the next $1/25$, the outermost $1/50$ of the ring in which it is contained. On the front surface the sectors represent similarly white areas of $1/50$, $1/100$ and $1/200$ of the corresponding black portion. The light difference tests are precisely similar, but the sectors here are black on white instead of white on black. A small ratchet rotator worked by thumb pressure is used for spinning the discs around.

7. When the light minimum disc is rotated the sectors appear as grayish rings on a black ground. The normal eye should distinguish all three rings on the front surface, even the outermost. The light minimum then equals $1/200$ or, as Percival more conveniently expresses it, 200. If a man can distinguish only the innermost ring, his LM is 50; if he can distinguish no ring on the front surface and only two on the back, his LM is 25.

8. A similar notation obtains for the LD disc, only here it is found that very few people can recognize more than two rings on the front surface of the disc, so that the normal LD may be stated as 100.

SOME CONTRIBUTIONS TO THE SCIENCE AND PRACTICE OF OPHTHALMOLOGY

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I. AN ILLUMINATED PERIMETER WITH CAMPIMETER FEATURES¹

This apparatus was devised in response to a request from a committee appointed by the American Ophthalmological Society to work out a better standardization of the illumination of perimeters and test charts. The request was for a feasible means of illuminating the perimeter arm with light of a good intensity and quality, so that every point on the arm in any meridian in which it may be placed shall receive equal intensities of light. Intensity and quality of illumination, however, are only two of the factors which influence the results of the perimetric determination. In devising the instrument described in this paper it has been our purpose to provide a control also of other factors which are of importance to the work of the office and clinic. The perimeter to be described in this paper has been adopted by the committee as the standard instrument for office and clinic work.

The variable factors which influence the apparent limits of color sensitivity are, so far as we have been able to discover, the wavelength and purity of the stimulus, the intensity of the stimulus and the visual angle, length of exposure of the eye, the method of exposure (moving or stationary stimulus), accuracy and steadiness of fixation, the intensity of the general illumination of the retina and its state of adaptation, breadth of pupil, and the brightness of the pre-exposure and of the background or surrounding field. The most important of these from the standpoint of the office or clinic are perhaps the intensity of the stimulus, the brightness of the pre-exposure and the surrounding field, the intensity of the general illumination, and the accuracy and steadiness of fixation.

Perhaps errors in refraction should be included in the above list of factors. They differ from those factors, however, in that they are a source only, or mainly at least, of differences in breadth of field between

¹ A description of the earliest model of this apparatus appeared in *Trans. Amer. Ophthal. Soc.*, 1920, 172.

observers, not for the same observer at different times. They are not, therefore, a serious source of trouble in the use of perimetry to check up the advance or recession of a given pathologic condition, but they are confusing in diagnosis. We have no certain means of telling, for example, how much of the results in any given case of high myopia is due to the refractive condition and how much if any is pathologic. To add to our knowledge on this point we are now conducting an investigation to determine the effect of various amounts and kinds of refractive errors on the results of field taking. We can not help but feel, as is stated and discussed later in the paper, that a provision should be made in our practice of field taking to include one set of results under correcting glasses, even though it means either working under conditions which give a narrow field or being content with results which do not include the wider portions of the temporal field.

1. INTENSITY OF STIMULUS.—By a sufficiently wide variation of this factor alone, the zones of color sensitivity may be made to have almost any breadth within the limits of the field of vision, to differ radically in shape, and even to change or reverse their order of ranking as to breadth,—changes of a type and magnitude to which the clinician might ascribe a grave and important pathologic significance. Without great precision in the control of intensity, it is obvious that reproducibility or result can not be obtained and little diagnostic significance can be attached to extent or shape of field, to order of ranking as to breadth of field, or to any variations from time to time or from person to person in these important features. When pigment surfaces are used as stimuli, the illumination of the perimeter arm determines the intensity of the stimulus light. Two methods are proposed for securing an even illumination of the stimulus at every point on the perimeter arm and of reproducing this illumination from time to time.

Method 1.—When the source of light is inlaid in the surface of the arm or its continuation, the illumination on this surface will be equal for approximately 180° on either side of the source. The value of this illumination at every point will be equal to the normal flux of light from the luminous surface divided by four times the square of the radius of curvature of the perimeter arm, or four times the square of the distance of the eye from the perimeter arm.¹ In a later publica-

¹ For a discussion of the principle by which an even illumination of the perimeter arm is secured by this method, the reader is referred to the Trans. Amer. Ophthal. Soc., 1920, 165–167.

tion the method will be elaborated and a perimeter constructed on this principle of illumination will be described. This type of device is open to the objections of being difficult of construction and wasteful of light.

Method 2.—When the source of light lies in the perpendicular to the plane of the perimeter arm at its center of curvature, it will be equi-

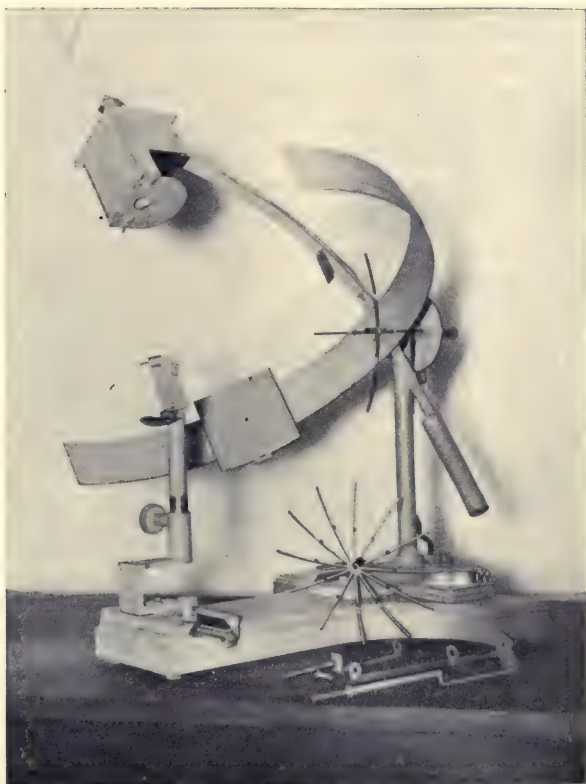


Fig. 1

distant from every point on the arm; also the angles of emission and incidence of the beam of light will be equal for every point on the arm. A perimeter (Figs. 1 and 2) has been constructed embodying this principle of illumination. Two arcs of the same radius of curvature were constructed at right angles to each other, one a 180° arc, the perimeter arm; the other a 90° arc, the lamp arm, at the end of which is

placed the source of light. In order that the source of light shall sustain a fixed relation to the perimeter arm for all positions of that arm, the two arms are fastened together at the center of rotation. About the source is a housing which is designed in such a way as to shield the eye of both patient and physician without interfering with the dis-

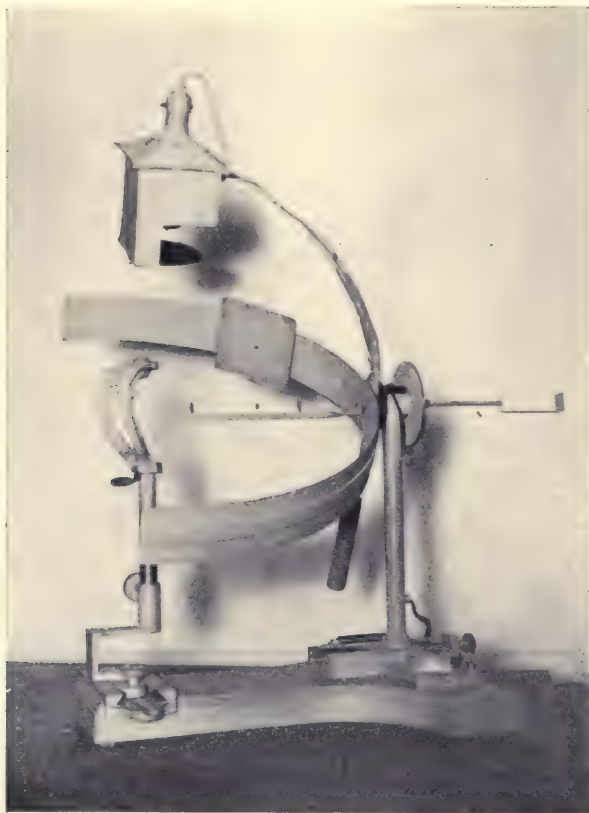


Fig. 2

tribution of the light which radiates freely from the filament to every point on the perimeter arm. The lamp house is well ventilated and the lower part is hinged to provide for the removal of the lamp. Provisions are further made in the construction of the lamp house for filtering the light to daylight quality. A well-seasoned type C₂ Mazda lamp operated by ammeter and rheostat control is used as the source

of light. The instrument is designed to run on any 110-volt circuit. This perimeter is not difficult to construct nor to operate. It provides for a uniform illumination of the perimeter arm in all meridians with light of a good intensity and quality; and with it a precision of control is possible which is comparable with the work of the physical laboratory. Of the two instruments we have constructed, it is without doubt much the more feasible and it is also very probably the more correct in actual practice. Both instruments are correct in theory.

2. THE BRIGHTNESS OF THE PRE-EXPOSURE AND THE SURROUNDING FIELD.—The brightness of the surface to which the eye is pre-exposed may change the apparent limits in certain meridians as much as 17° to 20° . A pre-exposure lighter than the color gives a dark, and one darker than the color a light, after-image. These after-images change profoundly the saturation of the color sensation, also its hue. A background or surrounding field lighter or darker than the color produces a similar effect on the limits, but not so great. In this case, the disturbing achromatic effect is due to physiologic induction or contrast. The variable effect of brightness of pre-exposure and surrounding field can be eliminated only by making both a gray of the same brightness as the stimulus color. Here again a precise control of the intensity of the illumination for all points on the perimeter arm becomes important. That is, the shade of gray which is needed to match the color in brightness changes with change of illumination; therefore, the selection of a gray which will match the color in brightness for all points of work presupposes constancy and uniformity of illumination. A further advantage is gained by making the background of the same brightness as the color. That is, when color and background are of the same brightness the stimulus disappears completely when the limit of sensitivity to that color is reached, instead of turning into a gray concerning the colorlessness of which the patient is apt to be in doubt. This gives the effect of the disappearance type of photometer and like it adds greatly to the ease and certainty of making the judgment.

For the control of brightness of pre-exposure and surrounding field cards are provided covered on one side respectively by grays of the brightness of the colors to be used as seen in the peripheral retina. At the center of each of these cards is pasted a disc of the appropriate color subtending a visual angle of 1° . To provide for the control of the pre-exposure for the stationary method of giving the stimulation, cards identical with the background cards are provided, covered also

on one side with a gray of the brightness of the color. The stimulation by this method is given as follows: The stimulus is placed at the point to be tested and covered with the pre-exposure card. The observer is told to take his fixation. At a given signal the stimulus is uncovered for one second and recovered. In case the moving stimulus method is used, the surrounding field serves as the pre-exposure.

3. THE ACCURACY AND STEADINESS OF FIXATION.—All are familiar with the disturbing effect of inaccuracy and unsteadiness of fixation. If correct and reproducible results are to be obtained, the eye must be accurately placed at the center of the sphere in the surface of which lies the perimeter arm, and the line of sight must not shift from the fixation point while the color observation is being made. As an aid to the correct placement of the eye and a check on its steadiness of fixation, two devices have been provided:

1. A small circular mirror is used as a fixation object in which the observer sees the image of his own eye.¹ When the eye is correctly placed with the line of sight normal to the surface of the mirror at its central point, the fact is indicated to the observer by the position of the image of his pupil and iris as seen in the mirror. Not only is this simple device of service in determining the correct position of the eye, but it aids the observer greatly in holding a steady fixation by giving him an immediate indication of deviations in the line of regard.

We scarcely need to point out that the ordinary fixation object does not afford an accurate control of fixation. Exact checking methods show that the observer is not always fixing the object when he thinks he is. The only guide to monocular fixation is clearness of seeing and this is a criterion that presents considerable latitude. However, with a mirror the observer has an objective check on the position of his eye.

One of the objections to the use of a mirror as a means of controlling fixation is the liability of glare from its surface, probably due to a combined specular and diffuse reflection rendering unnecessarily difficult the clear seeing of the eye by the observer. This objection has been obviated in our instrument by cutting off from the mirror the direct radiations from the lamp by a narrow shield which can be turned back out of the way when not in use. With the shield in position the eye receives from the mirror only the light which is first reflected from the eye to the mirror and then back to the eye. The elimination of the troublesome glare from the surface of the mirror is rendered particu-

¹ For a cut of the perimeter furnished with this fixation device, see *Trans. Amer. Ophthal. Soc.*, 1920, 167.

larly simple and easy in case of our instrument because of the plan of illumination employed, *i. e.*, the light all comes from a fixed source above and directly in front of the mirror.

2. The second device for the control of fixation is similar in principle to a peep-sight and may be called a parallax or peep-sight device. A small disc placed at the center of rotation of the perimeter arm is viewed through a circular opening in a small metal plate. The plane of both disc and opening is normal to the line of sight when the eye has the correct position and fixation. When the eye has this position and fixation the relation of size of disc and opening is such that the disc is seen not quite to fill the opening. The disc is painted black, also the edge of the opening, thus when the eye has the proper position and fixation, the edge of the opening is seen concentric to the disc with a narrow ring of the gray of the perimeter arm between. The control afforded by this device is very sensitive. A very slight deviation of the position or fixation of the eye results in the complete or partial extinction of this gray ring at a point in the direction of the deviation.

In order to locate the eye at the correct distance from the fixation object a light measuring rod 33 cm. in length is provided, to one end of which is fastened a small metal disc. In making the adjustment for distance one end of the rod is placed against the fixation object at its center and the distance of the perimeter from the observer's eye is changed by means of a coarse screw adjustment until the closed lid is just in contact with the metal disc.

A final important feature in the correct adjustment of the eye is to insure a constancy of relation of the meridians of the retina to the meridians of the field of vision as laid off by the perimeter arm; in other words, to guard against a slight tilting of the head to one side or the other. For this purpose we have designed a very small and unobjectionable mouth bit of light wood (Fig. 1) to be changed for each observer, so shaped that it can not be bitten too far forward or back, and thus the distance of the eye from the mirror be changed, or too far to one side or the other. There seems, however, to be an insuperable prejudice against the use of a mouth bit by both the physician and the patient. We have therefore designed a head rest (Fig. 2) which follows approximately the outlines of the forehead, side of the head and face, furnished with a suitably cupped chin rest the height of which is adjustable. To provide for individual differences in shape and breadth of forehead, an adjustable forehead piece or band of thin spring steel extending well around to the side of the head is screwed at

its central point to the forehead piece of the head rest. This forehead band is adjusted to fit foreheads of different shape and breadth by means of a set screw on either side near the two ends of the band. When the chin rest is adjusted to its proper height and the forehead band is made to fit the forehead, the patient's head is held comfortably in position and sufficiently rigid, it is believed, to satisfy the needs of office and clinic work. At least the probability of tilting the head to one side or the other, thus causing a disagreement of the meridians of the field of vision as indicated by the perimeter readings with the meridians of the retina is very greatly lessened, if not entirely obviated.

In order to quickly and conveniently locate the patient's eye at the center of the perimeter system three adjustments are provided: a rack and pinion to raise and lower the head, a second rack and pinion to shift the head to right or left, and a coarse screw adjustment to change the distance of the perimeter arm from the eye.

A very great practical need in a clinic perimeter is a method of controlling fixation for patients who have a central scotoma or pathologic blind area. With the eye properly adjusted for taking the fields these patients are not able to see a central fixation object. We have constructed a device for this purpose which can be inserted into the hollow axle of the perimeter in place of the mirror or the peep sight. This device consists of four light arms at right angles to each other curved to follow the arc of the perimeter arm, and of sufficient breadth to provide for all probable breadths of scotoma. Each of these arms carries a small stimulus, the distance of which from the center of the field is adjustable. In adjusting the patient's eye the physician looks through a small telescope contained in the hollow axle and lines up the pupil of the patient's eye with the cross hair in the field of the telescope. If desired the patient's eye can be made more visible by reflecting light from the lamp directly on it. This is provided for (Fig. 1) by placing a small oblong mirror of specular metal on the lamp arm at such a position and angle that the light received from the lamp will be reflected on the iris. This mirror is hinged to the lamp arm and can be turned back against it when not in use. When the eye is observed to be in position, the four stimuli are adjusted so that they can just be seen by the patient at the edges of the scotoma. These stimuli serve as the control of the patient's fixation, his instructions being so to direct the eye that all are visible.

This fixation device can be made serviceable for mapping the sco-

toma itself by adding 12 or more graduated arms equally spaced, provided with stimuli similar to those already described (Fig. 1). Then when the fixation is obtained by the adjustment of the four stimuli designed for that purpose, the further mapping of the scotoma is accomplished by moving the remaining 12 until they are on the edges of the scotoma. Or if preferred the physician may watch the patient's eye through the telescope directing the fixation by means of the cross hairs, while all of the stimuli are moved into position on the edges of the blind area. This objective control of the fixation by the physician may be used, if desired, both in mapping the scotoma or in taking the fields when a central scotoma is present.

Another important need in a clinic perimeter is a method of giving the correct location and fixation to eyes suffering with high myopia. Eyes with myopias ranging from 8 to 20 diopters would have great difficulty in seeing a fixation object at a distance of 33 cm. Because of the grave pathologic changes which take place in the retina and choroid of eyes suffering from high myopia, particularly in the region of the macula and nerve head, it is of great importance to be able to use both the perimeter and tangent screen in the examination of eyes in the more advanced stages of myopia. Three provisions have been made for this: (1) The mirror may be mounted on a rod sufficiently long to permit of its location at any point in the line of sight between the perimeter arm and the eye. This rod may be inserted into the tubular axle on which the perimeter arm rotates. (2) A peep-sight device is provided similar in principle to the one already described and so constructed that it may suffice as a fixation control for values of myopia ranging between 8 and 20 diopters. And (3) the perimeter arm may be illuminated with two intensities of light,—one carrying the fields well toward the periphery of the retina; the other giving limits narrow enough to fall within the corrected field of the glasses which are worn or may be worn by the patient. This feature provides also for the correction of high astigmatisms the presence of which make field taking annoying and uncertain as a diagnostic procedure. There are other advantages of providing for the taking of fields at more than one intensity of illumination: (a) Because of the concentric arrangement of the fibers in the nerve trunk and their order of distribution in the retina, it may be of importance as a point of diagnosis to sample the response of the retina at different degrees of eccentricity. And (b) the low illumination fields are in general more sensitive to the influence of the pathologic factors which cause the fields to have dif-

ferent breadths. This is in part due to the fact that low illumination fields are narrow fields. That is, sensitivity falls off gradually near the center of the retina, therefore, smaller changes of sensitivity are required near the center of the retina to expand or contract the field. It is probably also due in part to the change produced in the state of the retina's sensitivity at the low illumination.

The control of fixation for the presbyopic eye also presents a problem to the perimetrist. The eye with a high degree of presbyopia would have considerable difficulty in seeing with the necessary clearness a fixation object at a distance of 33 cm. By the use of the mirror as fixation control this distance is extended to 66 cm.; but the satisfactory use of the mirror requires that the image of the eye be seen fairly clearly. An eye without power of accommodation, even if there is no hyperopia for far seeing, is approximately 1.50 diopters out of focus for an object at a distance of 66 cm. When 1.50 diopters out of focus the eye can not see its image in the mirror with a satisfactory degree of clearness. However, the mirror can be used with a fair degree of satisfaction for lesser degrees of presbyopia.

We have three proposals to make for the control of fixation for the presbyopic eye: (1) The use of the mirror, if desired, for the lesser degrees of presbyopia. (2) The use of an illumination sufficiently low to bring the color fields within the field of the correcting glasses. And (3) the use of a peep-sight or parallax fixation device similar in principle to the one already described, with the target at a distance great enough to be seen by the eye without power of accommodation.¹ Sixty-six cm. has been chosen for this distance because (a) the target can be seen at 66 cm. with sufficient clearness to determine whether or not it is at the center of the viewing opening, formed by the hollow axle and tube, even though the eye is as much as 1.50 diopters out of focus; and (b) a greater distance presents difficulty as to feasibility of construction. The device is provided also with a lens the distance of which from the target can be varied from its focal length to that which is needed to render the target clearly visible at 66 cm. This lens is mounted in the end of a short tube which telescopes to the desired depth into the main tube at the end facing the target. By suitably changing the distance of the lens from the target the apparent distance of the target can be made to have any value between 6 m. and 66 cm.

¹ As already stated, the device for control of fixation in high degrees of myopia is also shown in Fig. 2 in position for use. When using either, the other should, of course, be removed.

By means of this optical device, therefore, a clear image of the target can be formed on the retina of patients having all possible degrees of presbyopia. However, as we have already stated, it is quite possible to secure a good control of fixation for any degree of presbyopia without the use of the optical attachment.

The steadiness of fixation is greatly influenced by the method of giving the stimulation. One of the serious objections to a moving stimulus is the difficulty of holding a steady fixation while the object to be observed is moving. The alternative procedure is the use of a stationary stimulus. That is, the stimulus is placed at the desired point on the perimeter arm and covered with the pre-exposure card. The observer takes his fixation and at a given signal the stimulus is exposed and recovered. By this method of giving the stimulation more time is consumed but a much greater precision of result is possible. A compromise procedure is recommended. That is, the approximate location of the limit is determined with the moving stimulus and the exact location with the stationary stimulus. By this compromise but very little more time is required and there is no sacrifice of precision.

In order to provide for the mapping of the normal blind spot and for the quick detection and mapping of central and paracentral scotomata, it has been deemed advisable to add to the perimeter a tangent screen, subtending a visual angle of 60 or more degrees. Provision is made so that this screen can be quickly and conveniently attached to the stimulus carriage and moved into position. The stimulus carriage and the tangent screen have at their exact center a circular opening equal in size to the cross-section of the tubular aperture about which the perimeter arm rotates. Thus when the tangent screen is in position, *i. e.*, with its central point in the axis of rotation of the perimeter arm, the tubular opening in the perimeter is continued through to the front surface of the tangent screen. This provides both for the exact adjustment of the tangent screen and permits of the convenient use with it of all of the fixation controls which we have described. Cards of white or black, as may be desired, with the fields laid off on the tangent scale are provided for the mapping of the area deficient in the light sense, and of grays of the brightness of the colors for mapping the color deficiencies.

In our own work we have found it convenient to use this large screen for a quick survey of the field for scotomata and a smaller screen similar to the one used to carry the colored stimulus in the field taking

for the actual detailed mapping of the scotomata and the normal blind spot. This screen was made considerably larger than the screens which serve as backgrounds for the colored stimulus in field taking in order that it might serve for the mapping of large scotomata and pathologically enlarged blind spots. It is mounted in a carriage of its own and is shaped to take the curvature of the perimeter arm. This screen is intended not as a substitute for the larger screen but as a supplement in cases in which such a supplement is found to be convenient and desirable. This device has the following advantages over the large central tangent screen for the actual mapping of the blind areas: (1) It can be moved to any part of the field from the center out to 90° in any meridian and its center located at the center of the area to be mapped. When the screen is properly centered the mapping can be done as it is on any tangent screen. (2) Upon each screen are drawn sixteen meridians radially from the center of the screen. These meridians are finely graduated so that the limits of sensitivity in any meridian can be read off for the permanent record and transferred to properly planned maps at the convenience of the experimenter. (3) Blind areas are most easily and precisely mapped when the stimulus is made to follow lines radiating from the center of the area. Unless there are such guiding lines it is difficult to pass from within out or from without in consistently when determining the limits of the blind area or when checking up the location of a limit by a second or third determination. On a large fixed screen these lines would have to be drawn specially for each scotoma. (4) All of the evidence points toward the importance of mapping the blind areas with colored stimuli—particularly the Mariotte spot. It is highly important that the determinations for the different colors be made on backgrounds of the same brightness as the colors, as will be shown by blind spot studies to be published later. It is much more feasible to arrange for this in case of the smaller movable fields than in case of a large fixed field. When the background for the smaller field is laid off in graduated radial lines it can serve for the mapping of many blind spots and scotomata before it need be replaced. In case of a large fixed screen, this superior adaptability and long service would not be possible. (5) With the movable smaller field it is more nearly possible to map all blind areas under the conditions of equal illumination which obtains in the perimetry of the color fields than it is in the use of the large fixed screen. In the latter case the illumination at which the mapping is done in one part of the field may be quite appreciably dif-

ferent from that at which it is done in another part of the field. The control of illumination, while not so important for mapping the blind areas to the light sense stimuli, is very important in the mapping of the blind areas to color.

With the controls provided in the perimeter recommended, a careful worker can without difficulty reproduce the limits of sensitivity within 1 or 2 degrees.

II. VARIABLE FACTORS WHICH INFLUENCE THE DETERMINATION OF THE COLOR FIELDS

We have already given a list of the variable factors which influence the apparent limits of color sensitivity and have stated that the most important of these factors from the standpoint of the work of the office and clinic are perhaps the intensity of the stimulus and the precision of its control, the brightness of the surrounding field and of the pre-exposure, the intensity of the general illumination, and the accuracy and steadiness of fixation. Space can be taken here for the discussion of the effect of intensity alone. Reference will, however, be given to discussions and data bearing on the other factors.¹

INTENSITY OF STIMULUS.—By a sufficiently wide variation of this factor alone the fields of color sensitivity may be made to have almost any breadth within the field of vision, to differ radically in shape, and even to change or reverse their order of ranking with regard to breadth. For example, with very high intensities the limits of red, yellow and blue are coincident with the limits of white light vision. Green can not be made to have so wide an extent. With stimuli of medium intensity and of the relative energies found in the prismatic spectrum of a Nernst filament the limits are concentric and in the order from widest to narrowest of red, yellow, blue and green. With stimuli of medium intensities of equal energy the limits of red, yellow and blue interlace or crisscross. The limits for green again are narrower. The limits for pigment stimuli may be either interlacing, or concentric in the order of widest to narrowest of red, blue and green;

¹ The Factors that Influence the Sensitivity of the Retina to Color. A Quantitative Study and Methods of Standardizing, *Psych. Rev. Monog.*, 1913, xv (1), 178; Factors Which Influence the Color Sensitivity of the Peripheral Retina, *Trans. Amer. Ophthal. Soc.*, 1920; The Extent and Shape of the Zones of Color Sensitivity in Relation to the Intensity of the Stimulus Light, *Amer. Jour. Physiol. Optics*, 1920, i, 185-213; The Limits of Color Sensitivity: Effect of Brightness of Pre-exposure and Surrounding Field, *Psych. Rev.*, 1920, xxvii, 377-398.

or of blue, red and green depending upon the pigments used and the intensity of light falling on the perimeter arm. It seems only fair to conclude, therefore, that the conventional clinic rating of the limits from widest to narrowest in the order of blue, red and green is a function of the relative and absolute intensity of the stimuli employed as well as of the actual distribution of sensitivities. A change in the intensity of the stimulus not only changes the limits but, because of the irregular distribution of sensitivities in the different meridians, causes a marked change in the shape of the fields; and because of changes in the ratio of sensitivity to the different colors in the same meridians, a change in their ranking as to breadth. Without great precision in the control of intensity, it is obvious that reproducibility of result can not be obtained and little significance can be attached to extent or shape of field, to order of ranking as to breadth of field, or to any variations from time to time or from person to person in these important features.

The effect of changes in the intensity of the stimulus both on the extent and shape of the color fields varies with the order of magnitude of intensity employed. For medium and low intensities the effect of a given amount of change is very much greater than for high intensities. This is an obvious corollary of the type of distribution of sensitivities found in the peripheral retina. That is, in passing from the center toward the periphery of the retina the decrease in sensitivity is comparatively slow and gradual in the paracentral retina; it is much faster and more abrupt in the mid periphery; and very abrupt in the far periphery. It requires, therefore, comparatively large changes in stimuli of high intensity which carry the limits of sensitivity into the far periphery, to produce a significant change in the fields; not so great a change in stimuli of medium intensity; and a still smaller change in stimuli of low intensity. This effect varies greatly, however, for the same color in the different meridians and for different colors in the same meridian. For stimuli of the medium and low intensities used in the office and clinic, the effect of change of intensity is very marked indeed both on the extent and shape of the fields of sensitivity.

We have stated that the order in which the fields may be found to occur ranked with regard to breadth depends both upon the actual distribution of sensitivities, and upon the relative and absolute intensities of the stimuli employed. That it depends upon the relative intensities of the stimuli employed should be more or less obvious, provided, of course, that the relative differences of intensity are great

enough to overcome or reverse actual differences in sensitivity. In support of the statement that it depends also on the absolute intensity, maps will be shown in which for the same eye and a constant ratio of intensity of stimuli, the limits for red and blue in some cases interlace or crisscross; in others they are concentric or nearly so, the limits for blue lying outside of the limits for red or conversely the limits for red lying outside of the limits for blue,—the difference in result depending solely upon the absolute intensities of the stimuli employed. The

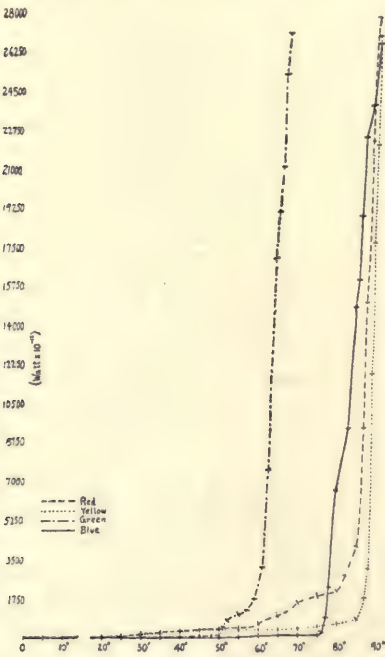


Fig. 3.—Chromatic thresholds for the four colors, temporal meridian.

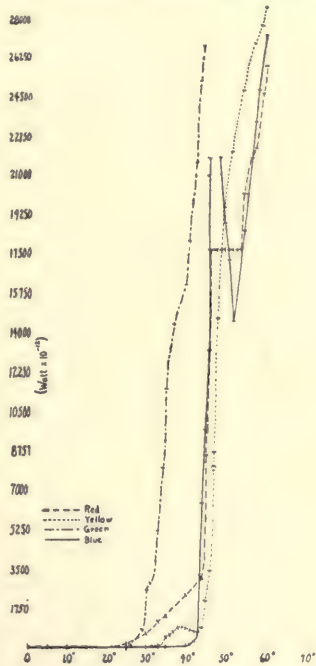


Fig. 4.—Chromatic thresholds for the four colors, nasal meridian.

determinations were made with the Hering standard pigments presented to the eye by means of the perimeter just described, with the pre-exposure and surrounding field of the brightness of the color, the only variable being the intensity of illumination of the perimeter arm.

The effect of such factors as intensity on the extent and shape of the color fields and their order of ranking as to breadth can be better understood when a thorough knowledge is had of the actual distribution of sensitivities from point to point from center to periphery of

the retina. In order to show this distribution of sensitivity from center to periphery of the retina we have made determinations of the threshold of sensation (the amount of light required just to arouse the color sensation) with spectrum lights and under proper conditions of control for the different colors at near lying points in the different meridians. A graphic representation of the results of these determinations for two meridians, the temporal and nasal, is given in Figs.

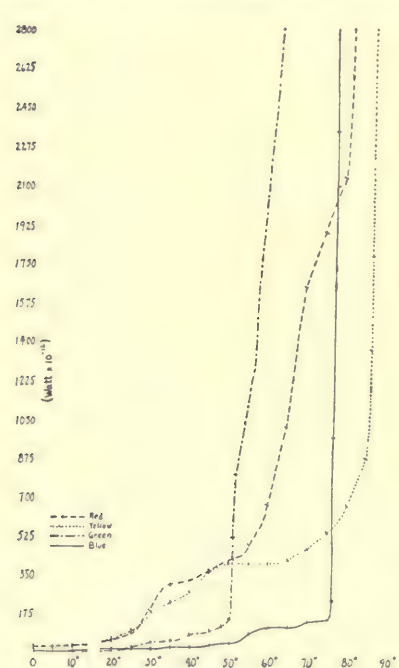


Fig. 5.—Chromatic thresholds (enlarged scale) for the four colors, temporal meridian.

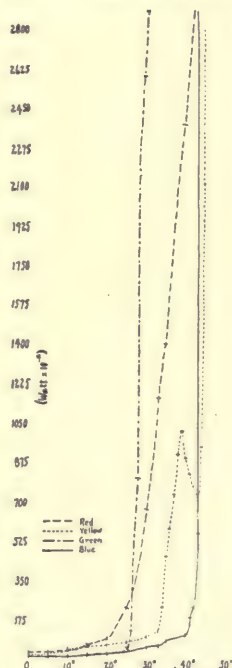


Fig. 6.—Chromatic thresholds (enlarged scale) for the four colors, nasal meridian.

3 to 6. In these curves the degree of eccentricity is plotted along the horizontal co-ordinate and the energy or intensity values of the threshold in watts (10^7 ergs per second) are plotted along the vertical co-ordinate. Maps have been made also showing the effect of varying the intensity of the stimulus on the breadth, shape, and order of ranking of the color fields as to breadth in sixteen meridians. Two sets of conditions have been used: high intensities, the fields having been taken with our rotary campimeter and spectrum lights; and medium

and low intensities, the fields having been taken with the perimeter described in the preceding section and pigment stimuli. Space will be taken here for a representation only of the results obtained under the second set of conditions.¹

In this second series of determinations five intensities of light reflected from pigment stimuli were used: the Hering standard pigments under 51, 17, 7, 3 and 0.03 foot-candles of light. In order to show how these illuminations compared with the day-light illumination falling on the perimeter arm in an ordinary room, measurements were made of the daylight at the level of the perimeter in a room with a window, southern exposure, the lower sill of which was somewhat higher than the level of the perimeter arm placed in the horizontal. These measurements were made on a bright day, January 14, 1921. It was found that our highest intensity of illumination, 51 foot-candles, was the same as that falling on the perimeter arm at the fixation point when facing the window at 1 P. M.; our second highest illumination, 17 f. c., was the same as that falling at the fixation point at 2.30 P. M.; the third highest illumination, 7 f. c., the same as that falling on the fixation point at 3.45 P. M.; and the fourth, 3 f. c., the same as that falling on the fixation point at 4.15 P. M. Measurements were also made with the perimeter placed parallel to the window. Our second illumination, 17 f. c., was the same as that falling on the fixation point at 11 A. M.; the 7 f. c. illumination, at 1.30 P. M.; and the 3 f. c. illumination, at 2.30 P. M.

The decrease of illumination from 51 to 3 foot-candles narrowed the limits for red by an amount ranging from 11 to 37 degrees; blue, 13 to 37 degrees; and green 10 to 19 degrees, a result, it will be remembered, which was produced by a change of illumination equal to that which occurred from 1 to 4.15 P. M. in a well-lighted room on a bright winter day. The decrease of illumination from 51 to 0.03 foot-candles narrowed the limits for red by an amount varying from 27 to 72 degrees; blue, 29 to 61 degrees; and green, 22 to 45 degrees (Figs. 7 to 13).

Since this perimeter, with its pigment stimuli different from those now in use in office and clinic work, and its standardization and control of factors, introduces a new situation in perimetry, it becomes

¹ For the results for the first set of conditions, the reader is referred to *The Extent and Shape of the Zones of Color Sensitivity in Relation to the Intensity of the Stimulus Light*, *Amer. Jour. Physiol. Optics*, 1920, i, 185-213; *Factors Which Influence the Color Sensitivity of the Peripheral Retina*, *Trans. Amer. Ophthal. Soc.*, 1920; *The Absolute Limits of Color Sensitivity and the Effect of Intensity of Light on the Apparent Limits*, *Psych. Rev.*, 1920, xxvii, 1-23.

necessary to determine a norm for the color fields under the new conditions to serve as a standard or basis for comparisons. This work is in

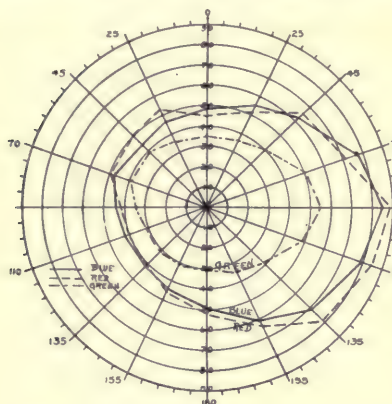


Fig. 7.—Fields taken under 51 foot-candles of light, an illumination equal to the daylight falling on the perimeter at the fixation point when facing a window on a bright day, 1 P. M.

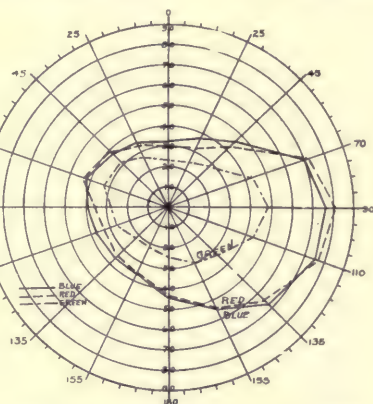


Fig. 8.—Fields taken under 17 foot-candles of light, an illumination equal to the daylight at the fixation point, perimeter facing a window on a bright day, 2.30 P. M., or at 11 A. M., perimeter parallel to window.

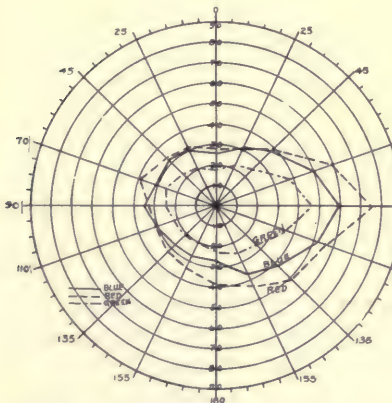


Fig. 9.—Fields taken under 7 foot-candles of light, and illumination equal to the daylight at the fixation point, perimeter facing a window on a bright day, at 3.45 P. M., or at 1.30 P. M., perimeter parallel to window.

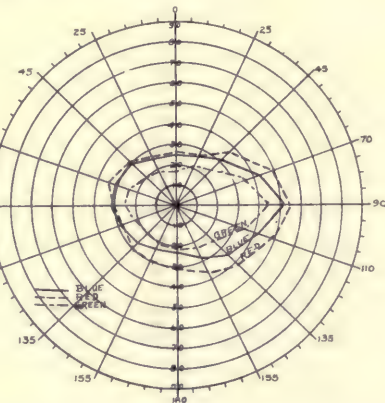
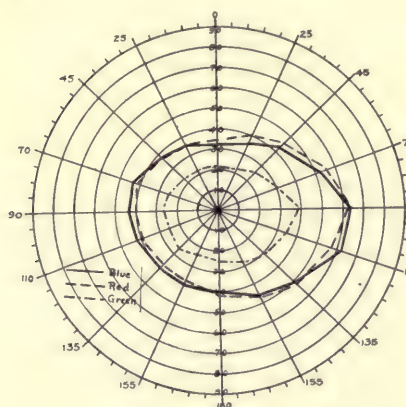


Fig. 10.—Fields taken under 3 foot-candles of light, an illumination equal to the daylight at the fixation point, perimeter facing a window on a bright day at 4.15 P. M., or at 2.30 P. M., perimeter parallel to window.

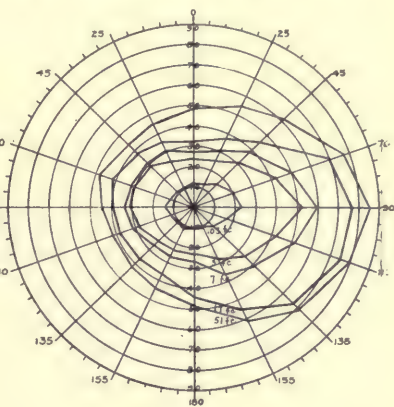
progress but has not yet been finished. Average fields for blue, red, and green have been determined for 35 normal observers just as the

perimeter stands, *i. e.*, with 17 foot-candles of light on the test object and with the controls described (Fig. 11). As fast as possible studies will be made also of typical pathologic cases,—also of normal and



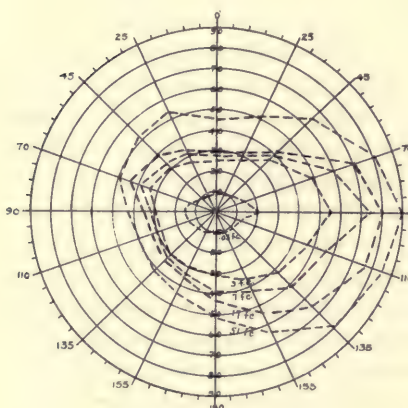
Average color fields

Fig. 11.—Fields for blue, red, and green, for 35 normal observers.

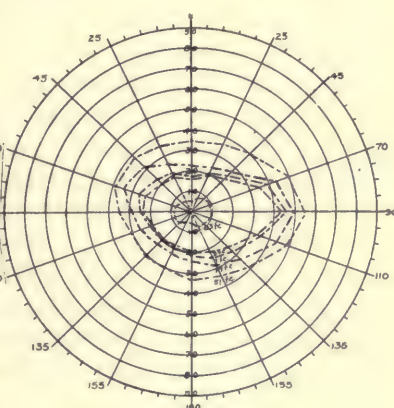


Fields for blue

Fig. 12.—Showing variations in both the extent and the shape of the color fields when taken under 51, 17, 7, 3, and 0.03 foot-candles of light.



Fields for red



Fields for green

Fig. 13.—Showing variations in both the extent and the shape of the color fields when taken under 51, 17, 7, 3, and 0.03 foot-candles of light

pathologic cases at a suitably selected lower illumination. An other feature of the advance work will be to ascertain how far if at all the determination of central sensitivity can be substituted for field taking

in the diagnosis of pathologic conditions; and how valuable as a supplement such determinations are in a diagnostic program. The quickness and the superior ease and precision with which the determination of central sensitivity can be made are strong points in favor of its use in practical work. A further advantage is that the determination can be readily made with the eye corrected for defects in refraction.

III. THE ACUITY LANTERN

This apparatus was devised in response to a request by the Eye Division of the U. S. Naval Hospital for a means of making a quick and accurate test of acuity at low illumination. Experience has shown, roughly speaking, that only 25 to 30 per cent. of the men on the battle-ships have a sufficiently keen acuity at low illuminations to qualify for all branches of the lookout and signal service work. The apparatus provides for a wide range of illumination in just noticeably different steps (beginning at 0.07 meter-candle or lower) with no change in the color value of the light and with a specification at each step of the intensity of light falling on the test-object.

For use in laboratory and clinic work the original apparatus has been elaborated to serve the following purposes: (a) for testing acuity with a means of varying, controlling and specifying the amounts of light used over a wide range of intensity of illumination; (b) for determining the exact amount and location of an astigmatism with great delicacy and precision and for using at will either the visual angle or the illumination scale for the detection of errors in refraction or in their correction; (c) for testing the light and color sense in terms of the amount of light required just to arouse the light and color sensations; and (d) for varying independently the saturation and brightness of a color and for color mixing.

Among the requirements for an apparatus for determining acuity at low illuminations or the effect of change of illumination, the following points may be mentioned: (1) A means of changing the illumination by small amounts over a wide range, beginning at or below the threshold for the test-object employed, without changing the color value of the light. If in making this change the color value of the light is altered it is obvious that another factor affecting the results is introduced. (2) A means of keeping constant for an indefinite length of time any desired intensity of illumination and of reproducing this intensity at will. (3) A means of specifying accurately at any point in

the scale the intensity of light falling on the test-object; and (4) it is desirable that the apparatus employed for controlling the illumination can be used with the test-objects already accepted in clinic practice.

The most difficult problem one has to face in constructing an apparatus for determining the minimum amount of light that permits of the discrimination of a given test-object, more particularly if that object consists of a line of test letters, is to secure a uniform illumination of the line. We were able satisfactorily to meet this difficulty in only one way, namely, by selecting an aperture sufficiently small to permit of its uniform illumination and projecting a magnified image of this aperture on the test card. That is, an aperture was selected of such a size and shape that when magnified five-fold it gave a band of light which just blocked off one line of the test letters. It is obvious that this aperture could be made of different sizes and shapes depending upon what is wanted in the projected image. For example, two or three lines of test letters could be blocked off if desired, or the whole card or any part of it could be illuminated, etc. There is no reason, moreover, why the aperture could not be made adjustable in size to suit the needs and preferences of the individual operator. In one model of the apparatus these apertures were cut in a series of slides which could be inserted in the projection tube just outside the lamp house in grooves in a light-tight boxing. A convenient means was thus provided for changing the aperture, if desired, during a series of tests without having to open the lamp house. The source of light is a well-seasoned Mazda C lamp of the round bulb or stereopticon type of 100, 250 or 500 watts, depending upon the range of illumination that is desired. The lamp is installed vertically in the roof of the lamp house at such a height that its filament is well above the aperture which is to be illuminated. In order to secure a uniform and diffuse illumination of the aperture the lamp house is lined with opal glass ground on one side. The aperture, 6 x 1 cm., is cut at the center of the cap covering the inner end of the projection tube. Further to aid in the even illumination of the aperture it is covered with a slide of ground glass. The changes in the intensity of light are produced by means of an iris diaphragm. When such a diaphragm is placed either at the front or back surface of the focusing lens, changes in the flux of light can be produced without any alteration in the size or the shape of the image produced by the lens, just as happens, for example, in the action of the iris of the eye. At a suitable point in the circum-

ference of the diaphragm is fastened a pointer which, as the diaphragm is opened and closed, moves over a translucent millimeter scale. This scale is mounted over a slot in the projection tube and receives its illumination from the light inside of the tube. The inside of the tube is painted a mat black. At the further end of the projection tube, 18.1 cm. from the illuminated aperture, in a brass ring and collar is mounted the focusing lens. This lens is 7.5 cm. in diameter and has a focal length of 14.8 cm. On the platform supporting the lamp house

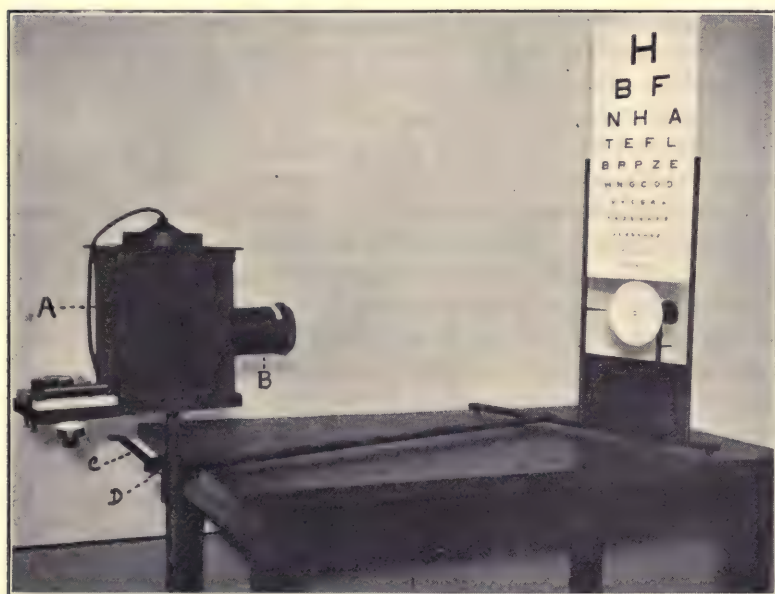


Fig. 14

are mounted a small Weston ammeter and a small rheostat to guard against fluctuations in the current and consequent fluctuations in light intensities. In order that any line of the chart may be illuminated at will, the lamp house is mounted on the end of a rod which is raised and lowered by means of a rack and pinion. The test card is mounted at a distance of 81 cm. from the focusing lens. A photograph of the apparatus is shown in Fig. 14.

In order that the intensity of light used at any time may be known, a calibration chart is provided in which are given the readings on the millimeter scale and the equivalent meter-candle values at the test

card. This calibration was accomplished as follows: The lamp house was removed and mounted on a photometer bar at a distance from the photometer head equal to its original distance from the test card. The scale was then gone over point by point and the meter-candle value of the light at the photometer head was measured. These values were corrected to conform at the center of the card to the cosine law.¹

For our own use in the laboratory we have preferred to substitute for the Snellen chart a single test character, the broken circle (the international test-object), which can be turned in different directions and the judgment of its direction rather than the recognition of the character be required of the observer as a test of discrimination. Our reasons for this preference are as follows: (1) A test letter may be recognized when it is not seen at all clearly. Recognition is too dependent on extraocular functions to be used with precision as a measure of ocular capacity. (2) The different letters of the Snellen chart set an unequal task for the resolving power of the eye. (3) An objective check is had on the judgment. This is especially helpful in case of children and the unintelligent, untrained, or subjective type of adult. (4) By the use of the same test character turned in different directions at will all possibility of learning the test series is eliminated. Also the test-object becomes more valuable for the detection of astigmatisms. And (5) at low illuminations the eye fatigues very rapidly. Thus if the task is the reading of the whole line of letters the results obtained measure not only acuity, but the power to sustain acuity which may or may not be compatible with the purpose of the test.

1. THE USE OF THE ILLUMINATION SCALE FOR THE DETECTION OF SMALL ERRORS IN REFRACTION AND IN THEIR CORRECTION.—There are doubtless many ways in which sensitivity can be added to the acuity test for the detection of small errors in refraction and in their correction. In connection with the problems which we have undertaken during the past ten years involving modifications and refinements in functional testing, three principles have come to light which can be used very effectively to this end: (1) An undue lag or slowness of discrimination and of making the adjustments needed for clear seeing. (2) A marked loss in power to sustain clear seeing. And (3) an increase in the amount of light required just to discriminate details in the standard acuity object. The devising of test methods based on

¹ For the calibration chart and the calibration curve in which the divisions of the millimeter scale are plotted against meter-candle values at the test card, see *Amer. Jour. Ophthal.*, May, 1920, iii.

the first two of these principles has been treated of in former papers. The third alone will be considered here.

The relation of the illumination scale to the detection of small errors in refraction and in their correction may be stated briefly as follows: Insofar as the test-object is concerned, clearness of seeing depends both upon the value of the visual angle subtended and the intensity of the illumination. It follows from this that either the illumination scale or the visual angle scale may be used for the detection of errors in refraction, *i. e.*, in the diagnostic procedure either the illumination may be held constant and the visual angle varied, or the converse. Since the visual angle scale sustains by convention a 1 : 1 relation to acuity while acuity changes slowly with change of illumination for all but very low illuminations, the illumination scale possesses the greater sensitivity for the detection of small errors in refraction—also the greater feasibility of contrivance and manipulation. Used in this way the illumination scale becomes in effect an amplifying scale—somewhat analogous to the use of the tangent scale in detecting small deflections in the magnet system of a galvanometer—and has an advantage in sensitivity in proportion to the amplification. In clinic practice it has been shown to be of particular value in determining the exact amount and placement of the correction of astigmatisms. That is, if the eye has equal resolving power in all meridians, the amount of light required just to discriminate the test-object in all meridians will be the same; if the resolving power is not equal, the amount of light required will be different in the different meridians and different in proportion to the amplification represented by the illumination scale. This gain in sensitivity over the clinic methods is needed in particular to determine the exact amount of the correction in case of high astigmatisms and both the amount and exact placement of the correction in case of low astigmatisms. The checking up of a number of cases shows that the corrections by the clinic methods may be and frequently are off from 0.12 to 0.25 diopter in the strength of the cylinder and, in case of low astigmatisms, from 5 to 20 degrees in the placement of the cylinder axis. While errors of this magnitude may or may not be troublesome in the ordinary uses of the eye—sometimes they are very troublesome indeed and perhaps always tend in time to increase the amount of the defect—they do constitute a much more serious handicap, in fact an actual disqualification, for work or vocations requiring special ocular proficiencies, *e. g.*, keen acuity, particularly keen acuity at low illuminations; speed in the use of the eye, especially

speed of discrimination and of making the adjustments needed for clear seeing; etc. Moreover a considerably greater amount of light is required as a comfortable and efficient working minimum by the poorly than by the well corrected eye. Indeed our experience with the tricornered relation of intensity of light, resolving power, and the retinal sensitivity to acuity has impressed us with the relative importance of resolving power in explaining the difference in the amount of light that is required by different people as a working minimum.

The relation of the intensity of illumination to acuity may be illustrated by the curve shown in Fig. 15. This curve represents the average results for four observers, tested by Koenig.¹ In this curve acuity is plotted along the ordinate and intensity of illumination along the abscissa. It will be noted, for example, that a change of from 1 to

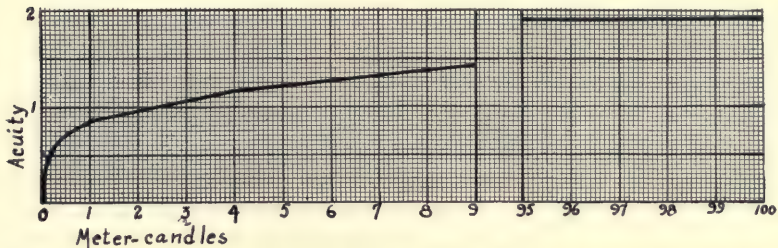


Fig. 15.—Showing the effect of increase in intensity of light on acuity (four observers), acuity plotted against meter-candles of light normal to the surface of test-object.

9 meter-candles, an increase of 800 per cent. in the intensity of illumination, produced an increase of only 74 per cent. in acuity; and a change from 9 to 100 meter-candles, an increase of 1011 per cent. in illumination, produced an increase of only 28 per cent. in acuity. The amplification within the latter range of illumination is doubtless too great for feasibility of application. That is, too wide a range of illumination would have to be used to compensate for the difference between the resolving power in the poorest and best meridians in the ordinary run of astigmatisms. The range from 1 to 9 meter-candles is, however, quite feasible and the relation between the two scales gives abundant sensitivity. These values fall within the range given by the apparatus described in the preceding chapter, 0.07 to 9.5 meter-candles. The testing of a large number of astigmatisms with this

¹ Ueber die Beziehung zwischen der Sehschärfe und der Beleuchtungsintensität, Verhändl. der Physikal. Ges. in Berlin, 1885, xvi, 79–83.

apparatus showed that in the majority of cases the minimum amount of light required for the discrimination of the opening in the broken circle (visual angle, 1 min.) in the most favorable meridian was of the order of 1 to 3 meter-candles; in the least favorable meridian, of the order of 6 to 9.5 meter-candles or more.

A very convenient apparatus for using the illumination scale for detecting low astigmatisms and small errors in the amount and placement of their correction has been described in the chapter immediately preceding. For this purpose the lantern was used to give the variations in illumination needed and the broken circle served as the test-object. The broken circle was fastened at the center of a graduated dial the opening of which (visual angle, 1 min.) could be turned into any meridian that was desired. The angle of turning could be read in terms of the divisions of the dial which was graduated to correspond to the readings on the trial frames used in office and clinic work.

Doubtless the apparatus can be used for the detection of astigmatisms in different ways depending upon the experience and preference of the operator. The quickest and most feasible method is first to make an approximate determination of the amount and placement of the correction by the clinic methods and employ the illumination method only for a more precise determination. In using this method as a refinement in the clinic methods, the procedure we ordinarily employ is as follows: The patient's eye is fitted with a cylinder of the strength and placement indicated by the clinic tests and the minimum amount of light required to discriminate the opening in the circle is determined in four positions, two in the meridian of the cylinder-axis and two in the meridian at right angles to this. If the minima are not equal in these four positions, the cylinder-axis is shifted and the determinations are made again, the four positions of the opening of the circle always being in the meridian of the cylinder-axis and the meridian at 90 degrees from it. If no placement of the cylinder is found which gives equal minima for the four positions, the strength of the cylinder is changed. The strength and placement of cylinder which requires both equal and the smallest amounts of light for the four positions of the test-object is accepted as the final correction.

In the testing and demonstration of the sensitivity and serviceability of the illumination method for determining the exact amount and placement of the correction of an astigmatism the following types of material have been selected: (1) Artificial astigmatisms made with cylinders of low diopter value. In choosing to include artificial astig-

matisms in this work it should be understood that we did not consider the artificial astigmatism the precise functional equivalent of the natural astigmatism. We are too strongly impressed with the possibility that the astigmatic eye may progressively acquire power to compensate in part for its defect to be of this opinion. They were selected because we wished to have in one set of cases an exact knowledge of the amount and location of the defect as a check on the determinations made by the test. (2) Natural astigmatisms without a cycloplegic. (3) Office and clinic cases with a cycloplegic. The difference in result between the most and least favorable meridians or between a true and false correction have thus far been of a considerably greater order of magnitude with than without a cycloplegic either in case of a natural or an artificial astigmatism. (4) Office and clinic cases in which the apparatus has been used merely to check up corrections already made by the clinic methods, objective and subjective. Among these cases it was comparatively rare to find one in which the minimum amount of light required to discriminate the test-object in the corrected meridian was equal or nearly equal to that required in the other meridians. Indeed in some cases the difference between the most and least favorable meridian exceeded the range of variation obtainable with the apparatus when provided with the 100-watt lamp. And (5) irregular astigmatisms.¹

In our own work we have found that the apparatus would be very helpful if it were used only to check up the corrections made by the clinic methods and were not employed further as an aid in finding out the exact amount and placement of the correction. For example, but very few minutes are required to determine with it whether any given correction equalizes or levels up the resolving power of the eye in the different meridians. The advantage of a checking method which is definite and at the same time feasible can readily be appreciated by any one who has tried to decide by the clinic methods in any wide range of cases just what should be the exact amount and placement of the correction of an astigmatism. The method has its chief value perhaps in those cases in which it is particularly difficult to make a decision by the clinic methods, that is, in determining the exact amount of the correction in case of high astigmatisms and both the

¹ For data showing the sensitivity of the method, see *Visual Acuity at Low Illumination and the Use of the Illumination Scale for the Detection of Small Errors in Refraction*, Amer. Jour. Ophthal., June, 1920, iii; *Sensitivity of Illumination Scale for Determining Exact Amount and Placement of Correction of Astigmatism*, *ibid.*, January, 1921, iv.

amount and placement of correction in case of low astigmatisms. The simple character of the judgment, namely, the mere indication of the direction in which the opening of the circle points instead of the more difficult task of deciding under the comparatively rough conditions of the office and clinic test whether this or that placement or strength of cylinder gives the clearer vision, together with the objective check on the correctness of each judgment, also contribute to make the method especially valuable in case of children, and the subjective, unintelligent or untrained type of adult. A further advantage of the method as worked out in connection with the present apparatus is its great sensitivity for the detection of irregular astigmatisms. The lack of satisfactory tests for this troublesome defect is generally conceded.

2. AN ATTACHMENT FOR TESTING THE LIGHT AND COLOR SENSE.—A consideration of the foundation principles of the acuity apparatus reveals at a glance that they lend themselves readily to light and color sense testing for clinic purposes. In order to convert the apparatus in the form described in this paper into a light sense tester three features are needed: (a) the choice of an aperture such that when magnified fivefold a stimulus is obtained of a size and shape suitable for a sensitive judgment of the threshold of sensation; (b) the provision of a suitable surface on which to project the magnified image of the aperture; and (c) means of reducing the intensity of light from the acuity threshold to the light sense threshold, *i. e.*, from the amount needed just to discriminate the standard acuity object to the amount needed just to arouse the light sensation. The iris diaphragm used in the present form of apparatus, range of pupil 5 to 65 mm., does not provide for this range of intensity without changing the source of light. It is obvious that an attachment for the further reduction of the light which does not interfere in any way with the use of the apparatus for the acuity work, would afford a more convenient means of securing the lower intensities than the changing of the source of light. Provision has been made for this in two ways: (a) by neutral absorption screens or filters; (b) by a Nicol prism (polarizer and analyzer); and (c) by a metal plate which may be made to move by minute amounts across the aperture of the iris diaphragm by a screw adjustment. The advantage of the two latter devices over the perishable filter is their very greatly superior constancy and permanency. The advantage of the moving plate over the Nicol prism is its extreme simplicity. The optical principle on which the use of the moving plate is based is that the image receives light equally from all parts of the

lens, therefore the transmission of light through any segment of the lens, whatever the size or shape of that segment, gives an equal distribution of light in the image. The attachment is made so that it will hold any of these reducing agencies, leaving the operator an option as to which shall be used. It is fastened to a narrow collar which slips over the end of the projection tube of the lantern and is held in place by a set-screw.

The testing of the color sense is provided for by inserting color filters in the beam of light. These filters are inserted in the attachment just described immediately in front of the lens and the intensity of light is cut down to threshold value by means of the iris diaphragm.

Color sense apparatus for clinic purposes seems at present, so far as the central field is concerned, to be limited to the testing of such gross deficiencies as are classed as color blindness. They are of little use for detecting the smaller changes which mark the advance and recession of many pathologic conditions. The present apparatus is designed for detecting and measuring the degree of deficiency in terms of the amount of light of a given range of wave-lengths which is required just to arouse the color sensation. It can be used, however, for testing color blindness in the conventional way by throwing the full strength of color on the test surface, or if a still higher intensity of light is wanted, by turning the lantern around to face the observer, instead of the test surface, and using the full strength of the beam. If desired, a ground glass plate can be inserted in the filter holder to diffuse the light and eliminate glare, or any strength of neutral filter to cut down the intensity. Also by means of the iris diaphragm the area of the colored surface may be varied from 5 to 65 mm.

3. AN ATTACHMENT FOR VARYING INDEPENDENTLY THE SATURATION AND BRIGHTNESS OF A COLOR AND FOR COLOR MIXING.—The attachment just described can be made into the form of a simple filter holder, or it can be made to serve the purposes noted above. We have constructed both types of attachment. The simple filter holder is made from three grooved metal strips 8 cm. long and of appropriate width and thickness, built into a three-sided rectangular figure open at the top. It is fastened to a narrow collar which slips over the end of the projection tube of the acuity lantern and is held in place by a set-screw. The more elaborate holder which can be made readily to serve the purposes of both attachments is constructed as follows: A frame holding one filter is made to travel from below in a groove across the aperture in the iris diaphragm by means of a screw motion. A second

frame holding a filter or opaque plate as may be desired, also operated by a screw, is made to travel from above in the same groove across the aperture in the iris diaphragm in the opposite direction. Each screw is furnished with a large head with a finely graduated beveled edge. As the screw is turned this head travels along a vertical mm. scale. Thus the position and movement of the filter can at any time be determined with great exactness and the scale be subjected to calibration in terms of amounts of colored and colorless light. The optical principle upon which this attachment is based is that the image receives equally light from every part of the lens. That is, when the colored filter covers a given segment of the lens aperture, the light transmitted is distributed uniformly through the image formed by the lens; or when an opaque plate covers a segment of the lens the light from the remaining segment is distributed equally in the image. Thus with the possibility of using one colored filter alone, one colored filter and an opaque plate to vary the amount of white or unfiltered light which mixes with the colored light, or two color filters, quite a wide range of variation can be produced in the composition and intensity of the light of which the image is formed. For example, (a) by use of one filter the color may be mixed with any amount of the available white light, *i. e.*, all the tints of the color may be produced from full saturation to white; (b) by the use of one filter and the opaque plate all degrees of saturation of the color at a constant brightness may be produced over a wide range of brightnesses and all the shades of the color from full saturation to black; and (c) by the use of two filters mixed colors can be produced with any proportion of the two components and of any brightness from full saturation to white.

IV. THE EFFECT OF VARYING THE INTENSITY AND COMPOSITION OF LIGHT ON ACUITY, SPEED OF DISCRIMINATION, SPEED OF ACCOMMODATION, AND OTHER IMPORTANT OCULAR FUNCTIONS

Space will be taken at this point only for an abstract of two articles bearing on the above topics.¹ One of our reasons for conducting studies of this type and for bringing them to the attention of the ophthalmologists is that we may enlarge our knowledge of the principles and possibilities of functional testing in relation to diagnosis and

¹The Effect of Variations in Intensity of Illumination on Functions of Importance to the Working Eye, Trans. of the Illuminating Engineering Society, 1920, xv, 769-801; The Effect of Variation of Visual Angle, and Intensity and Composition of Light on Important Ocular Functions, Trans. Illuminating Engineering Soc., Feb., 1922, xvii.

other important applications, and broaden our understanding of clear seeing and the factors upon which it depends.

In these articles the effect of increase of intensity of light is shown on the following functions: acuity, power to sustain acuity, speed of discrimination, and speed of adjustment of the eye for clear seeing at different distances. Wide ranges in change of illumination were used. The effect was found to be very much greater on the latter three than on the first of these functions. It was measured both on normal eyes and eyes with slight errors in refraction of a type and amount of frequent occurrence even in the corrected eye. The benefit of the increase was found to be considerably greater in case of these slight defects than for the normal eye. A comparison was made of the effect of increase of intensity of illumination and increase of size of visual angle. The importance of testing the neglected aspects of acuity: speed and power to sustain, in relation to diagnosis, vocational selection and hygiene or welfare work on the eye was demonstrated, and a comparison was made of their sensitivity and that of acuity and speed of adjustment, as test features for picking up small differences in the functional powers of the eye.

The investigation was also extended to include the effect of variations in the composition of light on acuity, power to sustain acuity and speed of discrimination. Two intensities of light at seven points in the spectrum were used, all made photometrically equal at each intensity. The highest acuity, speed of discrimination and power to sustain acuity were found in the mid-region of the spectrum (maximum in the yellow) even though, as is well known, lenses have their highest resolving power for the short wave-lengths. Again speed of discrimination and power to sustain acuity were much more affected by the change in the illumination condition (change of wave-length) than acuity. The relative importance of resolving power of the refracting media and resolving power of the retina is discussed in relation to the effect of changes in composition of light on acuity, speed of discrimination and power to sustain acuity.

Two comments are added: (1) All of the functions referred to above are aspects of acuity. They are aspects, however, which are not brought out by the conventional method of testing acuity. The conventional test of acuity takes little account of either speed or power to sustain, two aspects which are not only of great importance to the working efficiency of the eye, but are extremely sensitive indicators of differences in functional power, whether due to fatigue,

bad conditions of seeing, or refractive and pathologic disturbances. Add either of these aspects to the method of testing acuity and the effect is very similar, so far as sensitivity is concerned, to that obtained when an amplifier is added to a physical recording instrument. For example, changes of intensity of light which produce comparatively small differences in acuity, as ordinarily tested, cause very large changes in the speed of discrimination and the power to sustain acuity. The conventional acuity test is, comparatively speaking, not only insensitive, but it is not sufficiently comprehensive in the range of aspects covered to bring out differentially some of the most important functional powers of the eye.

(2) Considered with reference to the eye there are three factors in acuity or the power of the eye to see clearly—the resolving power of the refracting media, the space discrimination of the retina, and its sensitivity to light. To put it another way, there are the resolving power of the refracting media, or the power to form clear images on the retina; and the resolving power of the retina, or the power to discriminate detail in the physical image formed. Considered with reference to the stimulus light, six factors may be mentioned which are effective in acuity—the physical factors: intensity, wave-length, and purity of light; and their three subjective aspects: hue, saturation and brightness. Of these the wave-length and purity alone affect the resolving power of the refracting media or the power to form clear images. Intensity, hue, saturation and brightness affect the power to discriminate detail in these images, or what we have called the resolving power of the retina. For example, the benefit of the increase of intensity of illumination, cited above, came through the effect on the resolving power of the retina, not of the refracting media. Again the wave-lengths in the middle of the spectrum gave the highest acuity, speed of discrimination and power to sustain acuity although the resolving power of the refracting media is greater for the shorter wave-lengths. Here the effect of the more favorable hue and saturation of these colors (yellow, etc.) on the retina's power of discrimination more than compensated for the effect of wave-length on the resolving power of the refracting media. A third point is, the acuity and speed of the eye is greater for green light than for red; but the converse is true for the power to sustain. Doubtless the explanation here is that the refracting system has a higher resolving power for green than for red; but the retina sustains its resolving power better for the red than for the green, probably due in

part to the greater activity of the "streaming phenomenon" under the shorter wave-lengths, which is readily observed in the experiments on the power to sustain. A final striking example of the interplay of these two types of resolving power is the difference in the effect of wave-length on acuity in the microscopic field and in the visual field. In the microscopic field the shorter wave-lengths bring out detail more clearly, while in the visual field the longer yellow waves give the higher acuity. In the latter case the eye views its image directly; in the former, an image of the object formed by an accessory refracting system. That is, in the former case the relative importance of the refractive resolving power is greatly exaggerated. A discrepancy should be expected, therefore, between the effect of wave-length of light on acuity as represented in the natural eye and in the eye whose refracting powers are amplified by means of a microscope. In one case the resolving power of the retina is dominant; in the other the resolving power of the refracting system.

Both in administering to the welfare and comfort of the eye and in testing its powers analytically for diagnosis and other purposes, it seems important to know what factors influence the resolving power of the refracting media; what the resolving power of the retina; and, roughly speaking, in what proportions they combine to influence the total result in clear seeing.

V. THE ILLUMINATION OF TEST CHARTS

The question frequently comes before standardizing committees: At what intensity of illumination should acuity be tested? Our answer would be, It depends for what purpose the test is to be made. There are three obvious applications of acuity testing: vocational selection, diagnosis, and hygiene or welfare of the eye.

In the rating of eyes as to fitness for vocations, the test should be made as nearly as possible at the illumination usually employed in the vocation in question. The study of even a small number of cases shows that the eye can not be given the same relative rating as to acuity at different intensities of illumination. For example, experience has shown in the navy that only 25 to 30 per cent. of the men accepted for the service on the basis of the conventional acuity test at the higher illuminations are able to qualify for the lookout work at night on the bridge of the battleships. Further in a test of 61 observers made by us, all under 28 years of age and rating 6/4 acuity by the conventional test with 5 foot-candles of light on the test chart, 13 per cent. rated below 6/6 at 0.55 foot-candle of light and 33 per

cent. below 6/6 at 0.2 foot-candle. The acuity of the remainder was 6/6 or better at these illuminations. If speed in the use of the eye at low illuminations be added to the requirement, the scatter is very much greater still. The amount of time required just to discriminate

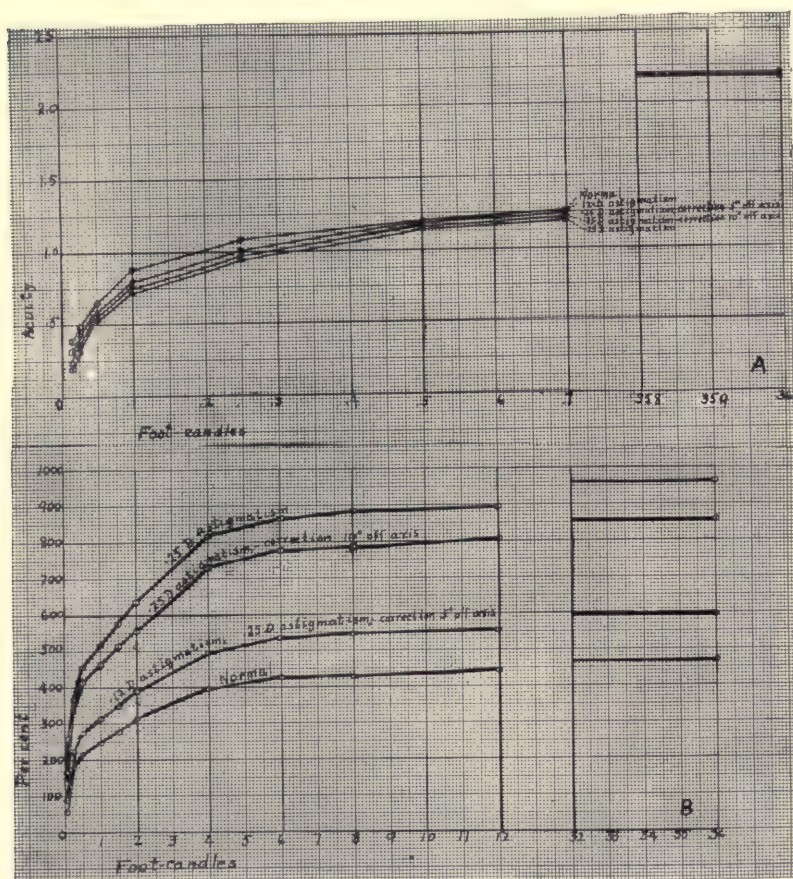


Fig. 16.—Showing the effect of increase of intensity of light on acuity for eye with normal refraction, and same eye made slightly astigmatic: A, Acuity plotted against foot-candles; B, percentage gain in acuity plotted against foot-candles.

1 minute of visual angle in this group of observers who were all put in the same class by the acuity test at the high illumination, covered a range from slowest to fastest of 1333 per cent. at 0.55 foot-candle and of 1443 per cent. at 0.2 foot-candle. It is quite obvious that any

attempt to rate eyes for vocational purposes at only one order of intensity of illumination is based on a lack of knowledge of the differential effect for different eyes of intensity of illumination on the power of the eye to see clearly.

All will agree, we suppose, that the object in diagnosis is to give the test under the conditions providing the maximum sensitivity for detecting errors in refraction. A glance at the curves given in Fig. 16 is sufficient to show that this maximum degree of sensitivity is not obtained at the higher illuminations. For the small uncorrected astigmatic errors represented in the chart the difference in acuity is scarcely detectable at the higher illuminations, but readily detectable at the lower. The reason for this is not hard to understand. The details in the slightly blurred astigmatic image can be discriminated at high illumination but not at low because of the effect of increase of intensity of illumination on the resolving power of the retina. This increase in diagnostic sensitivity was further directly tested out as follows: Low artificial astigmatisms were made and corrected. Starting with the proper placement of the correcting cylinder, the axis was shifted from its position by graded changes, ascending and descending series, until the judgment of just noticeable difference in the clearness of seeing of the letter B subtending a visual angle of 5 min. was made. This was done at 15, 10, 5, 3, 0.46 and 0.25 foot-candles of light on the test card. The tests were made very carefully. Seven concordant judgments out of 10 were accepted as the criterion of just noticeably different in any one set of trials. The results are shown in Table 1.

TABLE 1

Observer	Astigmatism	Degrees cylinder must be placed off axis to give just noticeable difference of clearness	
		High Illumination (Average of results at 5 and 10 f.c.)	Low Illumination (Average of results at 0.25 and 0.46 f.c.)
H.	0.25 cyl. ax. 90°	15.0	2.5
B.	0.25 cyl. ax. 90°	13.5	5.0
L.	0.25 cyl. ax. 180°	8.5	6.5
S.	0.37 cyl. ax. 180°	13.5	4.0
C.	0.50 cyl. ax. 90°	20.3	9.5
Bs.	0.75 cyl. ax. 90°	9.0	5.0

In any hygiene or welfare test of the favorableness of working conditions for the eye, the tests should also be made at more than one intensity of illumination. Conditions which are apparently acceptable at the higher illuminations are often far from equally acceptable at the lower illuminations. In all cases it will be found too that the sensitivity of the acuity test whether the purpose be vocational, diagnostic or hygienic, is very greatly enhanced when the procedure is made to include speed, power to sustain and accuracy, instead of accuracy alone as is the case in the conventional method of testing acuity.

DISCUSSION

DR. ALEXANDER DUANE (New York City): The paper of Drs. Ferree and Rand has many points of clinical value. One outstanding fact is the great variability of the illumination even in what seems like ordinary daylight and the corresponding variations in visual acuity thereby induced. Another is the effect of low illumination in accentuating the difficulties of vision produced by small refractive errors; and, as a corollary, the enhancement of an ability to detect these errors if we examine by reduced illumination. While such a reduction is effected most scientifically by the apparatus of the authors, it can also, as I have found, be accomplished in a rough-and-ready way by darkening the room or placing a dark glass before the eye tested; and these simple expedients actually do help us to come to a more certain decision in some instances where we are in doubt as to the strength or axis of a weak cylinder. As the authors point out, their observations show why some patients with very slight refractive errors demand a good illumination and suffer if they do not get it.

Another point most useful to the clinician is the fact that visual acuity as we measure it is a complex process, involving not simply the form sense but also the light sense and probably to some extent the color sense as well. Most enlightening is the fact brought out by the authors that in our tests we should be concerned not with visual acuity alone, but also with the power to sustain visual acuity, the speed of discrimination and the speed of adjustment. Failure in the first regard must conduce to asthenopia in spite of apparently normal vision and accurate correction, and failure in the others must render an eye unfit for certain of the finer functionings, *e. g.*, for work where the object of fixation is moving rapidly or for any other reason is changing its physical characteristics.

The observations on the great variation in the color fields produced by even slight changes in illumination have also an important clinical application. They should induce us either to attempt to standardize our color-field tests in some absolute and yet practical way, or else to cease ascribing to them any but a quite subordinate importance. To the latter view I have come after repeated experiences of the untrustworthiness of the tests as ordinarily applied.

Finally, the remarks on the use of the acuity lantern as a color mixer and as

a means of testing the lesser deficiencies of the color-sense indicate what may possibly be a very serviceable application of the instrument.

DR. LUTHER C. PETER (Philadelphia): We have watched with unusual interest the evolution of this perimeter. Conscious of the gross defects and of the inaccuracies of studies made on the extreme periphery by means of the perimeters at our disposal, the writer has largely abandoned a study of minute peripheral changes, depending either upon such qualitative and quantitative changes as one can determine on tangential planes for the detection of early peripheral loss. While this method also falls far short of the truth in slight contractions, a study of green and even red with low intensity stimuli at least established a normal for an individual patient with which subsequent examination could be compared, all at a minimum loss of time and with less trouble than when taken on ordinary perimeters.

To be able, however, to examine a peripheral field with a technique which in accuracy does not fall far below that of the physiologic laboratory, and to make such studies reproducible, has been the hope of the author. This hope promises well to be realized in the perimeter which Drs. Ferree and Rand have devised. Whatever criticisms may be offered, therefore, will be constructive or corrective from the clinical standpoint and not to detract from what we are all trying to obtain, namely, accuracy.

In the application of the instrument, the designers, in order to call attention to the varying results which may be obtained simply by varying degrees of illumination, the intensity of the stimuli, by pre-exposure and surrounding field, have clearly upset our beliefs as to the relative breadth of the several color fields. They have established authoritatively the fact that if the intensity of the stimuli is properly increased, the peripheral retina is sensitive to blue and red up to the limits of the form field, whereas green falls far short. Charts 3 to 6 graphically illustrate this well-established fact. It is important to observe, however, that the several colors require increasing degrees of intensity in order to make this condition possible. When we turn to clinical perimetry, conditions are different. Illumination, pre-exposure and surrounding field are the same as in establishing color thresholds, intensity of each color stimulus, however, remaining relatively the same from center to periphery. Red and blue fields under these conditions may interlace, they may be concentric, the blue lying without the red, or vice versa, the difference depending upon the absolute intensities of the stimuli.

The question naturally arises, cannot a uniform normal be established by varying the pigments so that interlacing in the normal individual can be eliminated? A study of the chromatic thresholds in Figs. 3 and 4 would lead one to think that this is possible. If this can be done, it will relieve a situation which is most confusing. By our present inaccurate methods blue is always found outside the red limits. If by more accurate methods, as made possible in this new perimeter, simply by varying the intensity of the pigments the same results may be obtained, why cannot a standard in pigments be fixed so as to eliminate interlacing in the normal? If studies in pathology are to give a true measure of pathological retinal sensitivity, the normal

should be made as definite as possible. Unless this can be done, our dilemma in determining what changes are due to pathological loss of sensitivity and what changes are due to faulty technique will only be carried further into the peripheral field. The same uncertainty, although to a more refined degree, will embarrass our studies.

By the suggested changes in technique made possible in a properly standardized perimeter such as the one presented, the principles of which can also be applied to tangent planes, red and blue in the normal are the only conflicting colors. If the writer has properly interpreted the paper under discussion, blue in the normal can be made to fall without or within the red limits by simply varying the intensity of the pigment stimuli. If this is correct, it would be most desirable to have the blue to fall regularly outside the red limits in normal conditions. This will continue to make our present limits of green, red, blue a fixed normal and will be a guide to a beginner to check up on his work in developing a satisfactory technique. It will do much more. There seems to be a definite relation between shrinkage of the blue limits and nutritional disturbances of the neuro-epithelium or the receptive part of the neuron. On the other hand, according to our studies in the past, red and green contraction seems to be greater than blue in disturbances of the transmitting part of the neuron or of the inner neuron and its axis cylinders. It is possible that this grouping of pathologic changes may undergo revision with better technique. A selection of the proper pigment, therefore, will help us to confirm our present beliefs, or convince us that we have been in error. Such a choice of the stimuli will also tend to clear up the perplexing problem of interlacing, especially of red and blue.

Without having had an opportunity to use this instrument in practical clinical work, it is only possible to speak in a speculative manner. In it are embodied the essentials of correct methods of measurement of the peripheral field under standard conditions. It promises much for those who are willing to give time to this method of study. To the group of men who really appreciate the refinements of careful diagnostic aids, it will be a welcome addition to office equipment.

DR. WILLIAM L. BENEDICT (Rochester, Minn.): Inasmuch as this paper was written for those who deal largely with diseased eyes in office and clinical work, it seems that we should judge of its value by the standards set for office and clinical work and not by the things that are found by the refinements of laboratory investigations. It is well known that we can vary the size of the field, that we can vary the acuity, and stimulate the fatiguability of the retina by changing the length of adaptation, the source of illumination, and the circumstances under which people are being tested. If this is possible with such variability for normal individuals, how much more must it apply to people who are undergoing tests under circumstances to which they are not accustomed. Individuals who are being subjected to routine examination for neurotic conditions find themselves more or less upset when they come to tests that require the co-operation that most of these tests of precision need. Individuals suffering from headache associated with brain tumor, those asso-

ciated with general metabolic disturbance we find in disease of the endocrine organs, are very uncertain as to their co-operation and as to the purpose for which the tests are being applied. So that there must be great uncertainty of results unless they are carefully interpreted by the man who makes the examination. It is important that the one who applies the tests apply them with a sense of appreciation of the delicacy of the individual being tested as well as the delicacy of the instrument.

I wish to say particularly in this regard that often patients worked with under such abnormal conditions respond with very great sameness to the ordinary rough tests. It is only by repeated examinations with tests for color fields with ordinary colors, when we get uniform fields day after day, that we are able in a long series of fields to work out the diminution of the light sense and of the field area to a degree which is sufficient to prognosticate the outcome of a rather serious neurotic lesion.

I wish to emphasize the importance of taking the color fields, particularly in the periphery, because some of our neuro-ophthalmic surgeons are beginning to cast doubt upon the importance of taking peripheral fields. It is important, it seems to me, that this be kept up, and that we urge as much as we can that accuracy of the fields in the periphery should be as important as accuracy of the fields in the central portion.

The particular things we are to receive from Dr. Ferree's laboratory work is the importance of getting the correct illumination at the side as well as in front, and we know we can do this with an ordinary perimeter if we will regulate our illumination. Then, with the individual watched as to his fixation, we can test with sufficient accuracy the visual fields so that we can urge on the surgeon the possibility of improvement by his type of treatment.

DR. WILLIAM ZENTMAYER (Philadelphia): As chairman of the committee of which Prof. Ferree was also a member, appointed by the American Ophthalmological Society to report on the Standardization of Test Cards and Perimeters, I feel that I should say a few words of appreciation of Prof. Ferree's work in connection with the illumination of the perimeter.

By the plan adopted the perplexing problem of having the arc of the perimeter equally and sufficiently illuminated in all meridians and at all points on the same meridian, has been solved. The method by which this has been accomplished is as simple as it is ingenious, and can be adapted to any of the perimeters based upon a revolving arc. He has added features which give to the instrument a much greater scientific value, as office tests can now be made under standardized conditions corresponding to those employed in the laboratory. Control of fixation, one of the most difficult factors in field taking, is effected in a practical way.

The campimeter attachment is a feature of worth and is superior to the hand campimeter in that it is uniformly illuminated and permits the examiner to keep the eye being tested under observation.

MR. J. GRAY CLEGG (Manchester, England): I feel under obligations to Dr. Ferree for the excellent instruments he has devised. They will be of

considerable assistance to the Council of British Ophthalmologists who are investigating some similar problems. From a practical point of view, however, one strongly desires a good perimeter to get reliable results for comparison, because atmospheric conditions vary and we have to examine patients both day and night.

I do not know whether Dr. Ferree has adopted this method, but I would like to ask whether the use, during these examinations, of an artificial daylight glass for illuminating the objects on the scotometer and perimeter is a really practical proposition for office work.

DR. C. E. FERREE (closing): In reply to Dr. Clegg I would say that the problem given to us by the Committee on the Illumination of Perimeters and Test Charts was to devise a means of illuminating the perimeter arm with light of a good intensity and quality so that every point on the arm in any meridian in which it may be placed shall receive equal intensities of light. Good quality means, of course, a close approximation to daylight. The satisfying of this specification has caused us considerable difficulty. A glass sufficiently blue to absorb the excess of the long wave-lengths present in artificial light lowers the total transmission so much as to make it difficult to get the intensity of illumination needed in perimetry with a lamp of a wattage feasible to use in type of the lamp house which has to be employed. In one of the instruments shown here the light passes from the lamp to the perimeter arm through a daylight cylinder or collar. The light so filtered is a close spectro-photometric approximation to north sky light but the total transmission is only 15 per cent. In the other instrument demonstrated the light filters through a double etched 75-watt, type C Mazda bulb. This bulb gives a higher transmission but only a rough approximation to daylight composition. With this lamp 17 foot-candles of light are incident on the perimeter arm. One objection to all blue bulb lamps is that the intense heat generated by the filament causes the glass to lose its color and to change its transmission. We will probably use finally a collar of approximately the same transmission as the type C bulb in connection with a clear bulb lamp. This collar will be far enough removed from the filament not to be affected seriously by the heat which it radiates.

In reply to Dr. Peter I would say that it would be quite possible to get pigment stimuli so graded as to reflection coefficient that the limits of the blue field would lie just outside the limits of the red field for the normal eye. Interlacing, which is the first step toward reversal, might then have a diagnostic value provided that the intensity of light on the perimeter arm were held constant. However, the exact composition of such pigments would have to be determined empirically, specified and carefully manufactured according to specification. It does not seem to me that enough advantage would be gained by the procedure to pay for the trouble involved. In fact, I doubt whether any advantage would be gained. With either the Hering or the Heidelberg pigment stimuli and the intensity of light used in our perimeter the limits for red and blue are interlacing for the normal eye. An amount of pathologic disturbance which would change the concentric fields

proposed by Dr. Peter to interlacing fields would change interlacing fields to concentric fields with red or blue on the outside, depending on whether the red or blue sensitivity was the more affected by the pathologic condition. Both procedures have their diagnostic features plainly marked and, so far as one can anticipate, there would be no difference in sensitivity between them. However, the clinician should be able to speak to the point of comparative diagnostic advantages better than I.

Dr. Benedict speaks of the ability to secure uniform results under ordinary daylight illumination. In our paper we show the variations obtained over a range of illumination made equal photometrically to that occurring from noon to four-thirty on a bright winter day in a well-lighted room. If one is to secure results reproducible within acceptable limits of precision under the vicissitudes of daylight illumination in this latitude, he will have to be very watchful indeed. It could be done with an illuminometer in a room properly curtained for control of intensity. Even then reproducible illumination is all that could be hoped for. A constant amount of light at every point on the arm for every position in which the arm might be placed would be scarcely possible to obtain. I surmise that Dr. Benedict means to urge us not to be too pessimistic about perimetry even under the unequal conditions afforded by daylight illumination. Perimetry done by a careful physician even under such conditions is very much better than no perimetry at all. I think that we would all be very glad to subscribe to this principle. However, I cannot urge too strongly the need for a precise control of the intensity of light falling on the perimeter arm. It is by far the most important control we have yet to attain in the general practice of perimetry. I say this without any wish to detract from the importance of the other variable factors the provisions for the control of which we have discussed and demonstrated. All are agreed, I believe, that what we especially need for diagnosis is constancy of conditions from day to day and from office to office, as scientifically correct and as sensitive as may be, in order that norms may be established and results widely compared as to the deviations found under the various pathologic conditions. When this is done, there is every reason to believe that much of the confusion that now exists as to results and their meaning will be cleared up and that field study will attain to the importance in the examination and diagnosis of eye diseases which its great sensitivity and fundamental relations entitle it to have.

LE DIAGNOSTIC ET LA MESURE DES VICES DE RÉFRACTION AU MOYEN DE LA FENTE STÉNOPÉIQUE ET DU CADRAN HORAIRE

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Le diagnostic et la mesure des vices de réfraction comporte plusieurs méthodes subjectives, parmi lesquelles celle de Donders est la plus vivement recommandée. Elle n'est pourtant pas à l'abri de toute critique. D'une façon générale, ses résultats, obtenus au prix de tâtonnements parfois excessifs, peuvent être erronés. En effet, l'évaluation de l'acuité visuelle maximum, sur laquelle est fondée cette méthode, dépend d'une appréciation, somme toute assez délicate, de nuances visuelles fort voisines. Il faut donc que le sujet examiné fasse preuve d'une certaine finesse d'intelligence et d'une attention soutenue, ce qui, on le sait, n'est pas toujours le cas. Dans l'astigmatisme, les difficultés sont encore accrues par l'emploi des verres cylindriques. Aussi n'utilise-t-on plus guère ces derniers que pour vérifier, après coup, les résultats objectifs de la skiascopie ou de la kératoscopie.

Ces inconvénients ont poussé certains ophtalmologistes à rechercher d'autres procédés dans lesquels l'examen subjectif des vices de réfraction ne fût pas intimement lié à celui de l'acuité visuelle. Les optomètres construits d'après les expériences de Scheiner représentent une des tentatives les plus intéressantes qui aient été faites dans cet ordre d'idées. Mais on s'accorde généralement à rejeter ces appareils de la pratique courante et à les considérer plutôt comme des instruments de laboratoire.¹

Dans un article publié dans les "Archives d'Ophtalmologie,"² nous avons montré quels services pouvait rendre, à ce même point de vue, une méthode basée sur l'emploi simultané de la fente stén-

¹ Voir Imbert, *Traité de Physique Biologique* de d'Arsonval, Tome II, page 620.

² *Diagnostic de l'astigmatisme au moyen de la fente sténopéique*, Arch. d'Ophtalmologie, mars, 1921.

opéique et du cadran horaire. C'est de cette méthode, de son principe, de sa technique et de ses avantages que nous allons nous occuper.¹

PRINCIPE DE LA METHODE

Abstraction faite de toute considération théorique, l'examen subjectif des amétropes, tel que nous le pratiquons, repose sur un fait très simple. Lorsqu'on encadre un méridien cornéen au moyen de la fente sténopéique, l'aspect du cadran horaire, vu à 5 metres, à travers le méridien ainsi isolé, varie selon la manière dont se comportent les rayons qui traversent ce méridien. Si ces rayons ont leur foyer sur la rétine (réfraction emmétrope), toutes les lignes du cadran sont vues avec la même netteté. Mais si leur foyer se trouve reporté en avant ou en arrière de la rétine (réfraction amétrope), l'aspect du cadran change et l'oeil ne distingue plus nettement qu'un seul diamètre (ou un groupe de 2 ou 3 diamètres immédiatement voisins): celui qui est orienté dans le sens du méridien encadré, tandis que les autres n'offrent qu'une apparence plus ou moins confuse. L'on se trouve ainsi en présence d'un phénomène optique comportant la production de deux images, susceptibles, à un moment donné, de se substituer l'une à l'autre.

Certaines de ses particularités doivent être bien mises en évidence.

¹ L'emploi de la fente sténopéique n'est pas nouveau. Quelques auteurs l'ont déjà signalé à propos de l'astigmatisme. Voici comment s'exprime le Docteur Ch. Abadie qui a été, à cet égard, l'un des plus explicites:

“Une fois la direction du méridien à corriger connue, un disque, percé d'une fente et monté sur un manche, sera placé devant l'oeil de telle façon que la fente du disque soit parallèle au méridien défectueux. De cette façon, la réfraction à travers tous les autres méridiens se trouve supprimée et l'oeil ne voit plus qu'à travers celui qui doit être corrigé . . . Pour déterminer la myopie ou l'hypermétropie de ce méridien, on essayera la série des verres convexes ou concaves jusqu'à ce qu'on ait obtenu le maximum d'acuité visuelle. Le numéro du verre auquel on s'arrête indique le degré d'amétropie du méridien. On aura, de la sorte, la direction d'un des méridiens principaux et le degré de son anomalie.

“De même, en plaçant la fente du disque dans une position perpendiculaire à la précédente, on déterminera la réfraction du deuxième méridien principal.”

(Traité des Maladies des Yeux, 1884, T. ii, page 376).

L'on voudra bien remarquer, après cette lecture: (1) Que le procédé, tel qu'il est décrit par le Dr. Abadie, est basé, lui aussi, sur la recherche de l'acuité visuelle maximum. Il en résulte que l'examen du sujet doit être pratiqué au moyen d'une échelle optométrique et non pas, comme nous le conseillons, d'un cadran horaire; (2) que, par suite, il n'est nullement question, dans ce procédé, de faire intervenir le phénomène optique qui sert de fondement à notre méthode—phénomène pourtant facile à mettre en évidence, mais qui ne semble pas avoir suffisamment attiré l'attention des ophtalmologistes au sujet des applications intéressantes auxquelles il pouvait donner lieu; (3) qu'enfin, et pour la même raison, la fente sténopéique, dans ce même procédé, est exclusivement réservée à l'étude de l'astigmatisme, tandis que l'emploi que nous en préconisons est général et s'applique aussi bien à l'astigmatisme qu'aux vices de réfraction du type sphérique.

Ces deux images possèdent des caractères parfaitement tranchés et sont facilement opposables l'une à l'autre; leur différence est assez manifeste pour qu'elle ne puisse échapper même aux sujets les plus bornés. En outre, le second aspect du cadran, par exemple, qui se produit tant que l'image de ce cadran ne se fait pas exactement sur la rétine, est remplacé par le premier d'une manière immédiate, brusquement pour ainsi dire (tout au moins en pratique), au moment où l'amétropie du méridien se trouve complètement corrigée. Ajoutons que ce phénomène optique se présente avec netteté même avec une acuité visuelle notablement réduite, pourvu cependant que celle-ci ne descende pas au dessous d'une certaine limite et que l'oeil puisse distinguer tout au moins la disposition générale des lignes du cadran.

L'application de ces données permet d'effectuer très simplement le diagnostic et la mesure d'un vice de réfraction quelconque, à la condition cependant qu'une distinction soit établie entre les amétropies du type sphérique et l'astigmatisme. Dans le premier cas, l'examen d'un seul méridien, pris au hasard, permet de déterminer la réfraction générale de l'oeil. Dans le second cas (il ne sera question ici que d'astigmatisme régulier), l'examen devra porter exclusivement sur les deux méridiens à réfraction extrême (méridiens principaux): ce qui ne peut se faire que si leur orientation est déjà connue. Il y aura donc lieu, chez les astigmates, de considérer un temps supplémentaire dans lequel on recherchera préalablement cette orientation.

TECHNIQUE DE LA MÉTHODE

Cette technique exige essentiellement le matériel suivant: (1) Un cadran horaire dont les lignes aient une certaine épaisseur (type du cadran de Green); (2) une monture d'essai double et dont la graduation soit identique à celle du cadran; (3) un disque noir percé d'une fente sténopéique et pouvant s'adapter sur la monture précédente;¹ (4) les séries convexe et concave des verres sphériques.²

¹ Nous conseillons de recourir aux lunettes d'essai et de rejeter, malgré tous leurs perfectionnements, les disques sténopéiques mobiles à manche ou à pied. Il est indispensable, en effet, si le sujet est astigmatique, que, pendant tout le cours de l'examen, la fente soit maintenue dans une direction rigoureusement parallèle à celle du méridien qu'elle délimite. C'est pour la même raison que nous conseillons de toujours placer le disque dans la rainure postérieure de la lunette; outre que la fente, grâce à son rapprochement de l'oeil, encadre plus étroitement le méridien examiné, on évite mieux ainsi son déplacement lors de l'emploi ultérieur des verres.

² On pourrait avoir recours à un optomètre du type Badal, muni d'une fente sténopéique et dont la photographie représentant l'échelle visuelle serait remplacée par celle d'un cadran horaire. Mais, pour la raison exposée dans la note précédente, son emploi dans l'astigmatisme pourrait donner lieu à des erreurs.

On examine chaque oeil séparément. Le sujet étant placé à 5 mètres du cadran horaire, on l'invite à définir l'aspect de ce cadran vu à l'oeil nu. Trois cas se présentent, selon que l'oeil examiné: (1) Ou bien distingue nettement toutes les lignes du cadran; (2) ou bien ne distingue nettement qu'une seule de ces lignes ou un petit groupe de lignes immédiatement voisines (pour la commodité de la description, nous désignerons cet aspect du cadran sous la dénomination de "phénomène de la ligne noire"); (3) ou bien ne distingue nettement aucune des lignes du cadran.

La technique se modifie légèrement suivant le cas considéré. Nous l'étudierons, en supposant, une fois pour toutes: (1) Que l'oeil examiné présente une intégrité absolue de ses milieux transparents et de ses éléments sensoriels; (2) que le muscle ciliaire possède toute son activité fonctionnelle.

PREMIER CAS—LE SUJET DISTINGUE NETTEMENT TOUTES LES LIGNES DU CADRAN.—L'oeil examiné n'est pas astigmat. Il peut être emmétrope, myope de très faible degré ou hypermétrope accommodant pour l'infini. Nous envisagerons successivement: (1) L'encadrement de cet oeil par la fente sténopéique; (2) le diagnostic de son état de réfraction; (3) la mesure, s'il y a lieu, de son degré d'amétropie.

1. *Mise en Place de la Fente Sténopéique.*—L'oeil est muni de la monture d'essai, sur la rainure postérieure de laquelle on adapte le disque sténopéique. La fente peut être disposée suivant une orientation quelconque; tous les méridiens possédant le même pouvoir réfringent, l'on n'a, en effet, à examiner ici qu'un seul méridien, pris au hasard.

2. *Diagnostic de l'Etat de Réfraction.*—Il résulte de l'aspect que prend le cadran après l'application du disque sténopéique.

Ou bien son aspect ne change pas, c'est à dire que toutes les lignes du cadran continuent à être perçues avec la même netteté. Dans ce cas, l'oeil est emmétrope ou hypermétrope. Plaçons devant la fente (dans la rainure antérieure de la lunette), un verre convexe de faible puissance. Si l'aspect du cadran reste le même, c'est que l'oeil est hypermétrope; dans le cas contraire, si le phénomène de la ligne noire se manifeste, l'oeil est emmétrope.

Ou bien l'aspect du cadran se modifie: le phénomène de la ligne noire apparaît. Dans ce cas, l'oeil est myope.

3. *Mesure du Degré d'Amétropie.*—L'oeil est myope—L'application de la fente a fait apparaître le phénomène la ligne noire.

Plaçons, devant la fente, des verres concaves de degré croissant. Le verre le plus faible qui provoque la disparition du phénomène, en rendant également nettes toutes les lignes du cadran, mesure le degré de la myopie.

L'œil est hypermétrope.—L'application du disque n'a pas modifié l'aspect du cadran vu à l'œil nu. Faisons passer, devant la fente, des verres convexes de degré croissant. L'un de ces verres provoque l'apparition de la ligne noire; celui qui le précède immédiatement mesure le degré de l'hypermétropie.

DEUXIÈME CAS—LE SUJET DISTINGUE NETTEMENT UNE SEULE LIGNE DU CADRAN OU UN GROUPE DE LIGNES IMMÉDIATEMENT VOISINES.—L'œil est astigmat. Son diagnostic devra se faire exclusivement suivant les deux méridiens à réfraction extrême. Ces deux méridiens seront examinés successivement et, pour chacun d'eux, on déterminera: (1) Son orientation; (2) son isolement au moyen de la fente sténopéique; (3) son état de réfraction; (4) s'il y a lieu, le degré de son amétropie. L'examen du premier méridien servira de type à notre description.

1. *Orientation du Méridien.*—L'astigmat, placé en face du cadran, ne perçoit nettement qu'une seule des lignes qui le composent, parfois un groupe de lignes immédiatement voisines (phénomène de la ligne noire). Or, la direction de cette ligne se trouve être précisément celle d'un méridien principal. On la relève, en lisant, sur le cadran, le degré qui lui correspond.

2. *Isolement du Méridien.*—L'œil étant muni de la monture d'essai, on dispose le disque dans sa rainure postérieure, de telle façon que la fente soit exactement orientée suivant le degré indiqué;¹ dès lors, on veillera, pendant toute la durée de l'examen, à ce que cette direction soit rigoureusement maintenue.

3. *État de Réfraction du Méridien.*—Le méridien, ainsi isolé, peut être emmétrope, myope ou hypermétrope accommodant pour l'infini. Deux cas se présentent.

Ou bien, l'aspect du cadran, vu à travers la fente sténopéique, ne change pas, c'est à dire que le phénomène de la ligne noire persiste; le méridien est myope.

Ou bien, cet aspect change; le phénomène de la ligne noire disparaît et toutes les lignes du cadran sont vues avec la même netteté:

¹ Si l'on avait affaire à un groupe de deux lignes noires, la fente serait orientée suivant la bissectrice de l'angle formé par ces deux lignes; si ce groupe comprenait trois lignes, son orientation serait celle de la ligne intermédiaire.

le méridien est emmétrope ou hypermétrope. Disposons, devant le disque, un verre convexe de faible puissance. Si le phénomène de la ligne noire réapparaît, on a affaire à un méridien emmétrope; sinon, si toutes les lignes persistent avec une égale netteté, le méridien est hypermétrope.

4. *Degré d'Amétropie du Méridien.*—Considérons successivement le cas d'un méridien myope et celui d'un méridien hypermétrope:

Cas d'un méridien myope—La fente sténopéique a laissé persister le phénomène de la ligne noire apparu à l'oeil nu. Faisons passer, devant elle, des verres concaves de degré croissant. Le verre le plus faible qui provoque la disparition du phénomène et rend toutes les lignes du cadran également distinctes, mesure le degré de myopie du méridien.

Cas d'un méridien hypermétrope.—La fente sténopéique a fait disparaître le phénomène de la ligne noire. Plaçons, devant cette fente, des verres convexes de degré croissant. L'un d'eux provoque la réapparition du phénomène. Celui qui le précède immédiatement mesure le degré d'hypermétropie du méridien.

Nous serons plus bref en ce qui concerne l'examen du second méridien. La fente sera disposée suivant une direction exactement perpendiculaire à celle qu'elle occupait précédemment (les deux méridiens principaux étant, comme on le sait, perpendiculaires entre eux). L'aspect du cadran, examiné à travers la fente ainsi orientée, nous permettra, grâce aux notions qui viennent d'être exposées, de diagnostiquer l'état de réfraction du second méridien et de mesurer, s'il y a lieu, son degré d'amétropie.

Comme on le voit, l'emploi de la fente sténopéique et du cadran horaire nous donne le moyen de mettre en évidence l'un des éléments indispensables au diagnostic de l'astigmatisme: la puissance dioptrique des deux méridiens principaux. Dès lors, connaissant la direction de ces méridiens, on peut établir la formule nécessaire à la prescription des verres correcteurs.

TROISIÈME CAS—LE SUJET NE DISTINGUE NETTEMENT AUCUNE DES LIGNES DU CADRAN.—Il s'agit, dans ce cas, soit de myopie, soit d'hypermétropie accompagnée d'un déficit plus ou moins marqué de l'accommodation pour l'infini, l'une ou l'autre pouvant être simple ou compliquée d'astigmatisme. Nous aurons à diagnostiquer: (1) La nature de l'amétropie; (2) son degré.

1. *Nature de l'Amétropie.*—On la détermine avec le seul secours du cadran horaire. Plaçons, devant l'oeil examiné, tout d'abord les

premiers verres de la série concave. Si les lignes du cadran deviennent toutes également plus distinctes, on a affaire à de la myopie simple; si l'une seulement de ces lignes devient plus nette, c'est qu'il s'agit d'astigmatisme composé myopique (méridiens principaux tous deux myopes); l'on note soigneusement alors la direction de cette ligne. Les verres concaves n'améliorent-ils pas la vision, l'on procédera de la même manière avec les verres de la série convexe; ceux-ci nous feront savoir s'il s'agit d'hypermétropie simple ou d'astigmatisme composé hypermétropique (méridiens principaux tous deux hypermétropes).—Il est préférable de commencer par les verres concaves, car les formes myopiques sont les plus fréquentes.

2. *Degré de l'Amétropie.*—Sa détermination nécessite l'intervention du disque sténopéique. La mise en place de la fente diffère selon que l'on est en présence d'une amétropie du type sphérique ou d'astigmatisme. Dans le premier cas, la fente encadrera un méridien quelconque, pris au hasard; dans le second cas, il faudra la disposer successivement suivant les deux méridiens principaux.

Le méridien ainsi encadré est, nous venons de le voir, myope ou hypermétrope avec accommodation déficiente pour l'infini. Quelle que soit la nature de son amétropie, la simple application du disque au devant de la cornée détermine l'apparition du phénomène de la ligne noire.¹ Le changement que subit cet aspect du cadran sous l'influence de verres sphériques surajoutés permet de mesurer le degré d'amétropie du méridien.

Cas d'un méridien myope.—Ici encore, le degré de myopie est déterminé par le verre concave le plus faible qui fait disparaître le phénomène de la ligne noire en rendant toutes les lignes également nettes.

Cas d'un méridien hypermétrope.—Faisons passer, devant la fente sténopéique, des verres convexes de degré croissant. A un moment donné, par suite d'une correction partielle de l'hypermétropie, l'accommodation produit son plein effet: Le phénomène de la ligne noire disparaît et toutes les lignes sont perçues avec la même netteté. Continuons à interposer des verres de degré de plus en plus élevé; l'un d'eux fera réapparaître le phénomène; le verre qui le précède donnera le degré d'hypermétropie cherché.

¹ L'apparition de cette ligne est immédiate si le degré d'amétropie n'est pas trop élevé et permet d'entrevoir tout au moins la disposition générale des lignes du cadran; dans le cas contraire, le phénomène n'apparaît qu'après interposition d'un verre corrigeant partiellement cette amétropie.

AVANTAGES DE LA MÉTHODE

Cette méthode comporte plusieurs avantages :

1. Elle n'exige aucun outillage nouveau. Bien plus, elle simplifie l'outillage existant, grâce à la suppression des verres cylindriques et parce que le cadran horaire est susceptible d'un usage universel, convenant aussi bien aux lettrés qu'aux illettrés et à ceux dont l'écriture ne correspond pas au type courant (Arabes, Russes . . .).

2. En raison de la simplicité et de la netteté du phénomène optique qui la caractérise, elle permet un examen rapide et précis des amétropies, quels que soient l'état de la réfraction et l'intelligence du sujet à examiner.¹

3. Sa supériorité éclate spécialement à propos de l'astigmatisme. A l'encontre de la méthode des verres cylindriques, nous avons affaire ici à une méthode permettant de déterminer, avec facilité et de prime abord, ce vice de réfraction.²

4. Pour conclure, nous dirons que cette méthode, en ce qui concerne le diagnostic et la mesure des amétropies, est plus précise et moins malaisée à appliquer que la méthode de Donders.³ Nous n'hésitons pas, en outre, à affirmer que, sauf pour certains cas très particuliers (simulation, enfants trop jeunes, diminution trop considérable de l'acuité visuelle . . .), cette méthode peut soutenir la comparaison avec la plupart des méthodes objectives. C'est ainsi qu'elle peut rendre les plus grands services à tous ceux qui ne sont pas familiarisés avec les difficultés de la skiascopie ou qui, momentanément, ne peuvent disposer d'une installation appropriée à ce dernier mode d'investigation (armées en campagne, tournées ophtalmologiques dans le bled).

¹ L'existence d'une contraction accommodative, fréquente chez les jeunes sujets, peut être une cause d'erreur à laquelle on remédiera, ici aussi, par des instillations d'atropine.

² Ce qui rend si compliquée la détermination de l'astigmatisme par la seule méthode des verres cylindriques, c'est que, les différents méridiens de la cornée laissant simultanément passer les rayons lumineux, l'oeil, au cours du même examen, peut accommoder tantôt pour l'un, tantôt pour l'autre des méridiens principaux. Ici, par contre, du seul fait que les rayons ne traversent à la fois qu'un seul méridien, cette intervention désordonnée de l'accommodation se trouve supprimée.

³ Elle ne saurait toutefois pas la remplacer complètement. A la méthode de Donders est réservé, en effet, un rôle fort important et qui lui est propre. Complément obligatoire des autres méthodes, elle intervient à titre de contrôle dès que l'on aura effectué, à l'aide de ces dernières, le diagnostic et la mesure du vice de réfraction. Elle nous renseigne alors sur l'acuité visuelle de l'oeil exactement corrigé et nous fait connaître le verre le mieux toléré et le plus utile. Elle permet ainsi une prescription plus appropriée à l'état physiologique du sujet.

ANOMALIES OF OCULAR DEVELOPMENT AND PIGMENTATION

J. GRAY CLEGG, M.D., B.S., F.R.C.S.

Manchester, England

BILATERAL SYMMETRIC CONGENITAL CORECTOPIA WITH IRIDODONESIS AND MICROCORIA; MICROLENTIS AND COLOBOMA LENTIS

William S., male, aged fifty-eight.

History.—Always poor sight. Worked as stoker and engine tender. Could not see enough to gain much at school. One half-brother and two half-sisters, all by one mother. All have good sight except the brother, who is short-sighted and wears glasses. No other member of family had eye trouble except one with squint. Patient has five daughters and one son. All have normal eyes. Patient is of impression that when a child he was brought to the Manchester Royal Eye Hospital and was told sight was wrong way up.

Sight got better as grew older until forty-six or so, but since then worse. Could just read newspaper with the left eye by holding near face and screwing eyelids up. Could only see headings of newspaper with the right. Never could obtain glasses to help.

Came under my care about June, 1921, because he frequently ran against people in street.

Condition on Admission, September 27, 1921: Extrinsic muscles act well. Lids, conjunctivæ, and lacrimal passages normal. Cornea clear, but a trifle smaller than normal. Diameter, 10 mm. vertically by 11 mm. horizontally. Anterior chambers rather deep. Irides and pupils as in sketch. Plane of irides flat and coronal. The tissue of the iris thin but thickened in immediate neighborhood of the pupils. Pupils reacted definitely to light. Drugs had no marked effect on the pupils, except that atropin alone brought about some dilatation, increasing the pupillary area to double its ordinary size.

It was impossible to see through the pupils with the ophthalmoscope, but they lighted up well by transillumination, showing as small, irregular sided slits, the pupillary margins of the iris roughly dovetailing their prominence and fissures when contracting.

With a strong loupe the stretched iris tissue appears as irregular alternating bands of blue and brown, but the tissue surrounding the pupil is finely radially furrowed, and of a light brown tint. V.R.E. = fingers; V.L.E. + 2 sph. = J. 20.

October 13th: V.R.E. + 3 D. sph. = J. 20; V.L.E., 6/60; with + 3 D. sph. = J. 20.

October 26th: Iridectomy inner side (Fig. 1).

October 29th: Lids swollen, good hole in iris, somewhat square in shape.

November 2d: Lens appeared in anterior chamber completely opaque. Calcareous, with small coloboma below.

November 3d: Lens slipped back into vitreous cavity; could not be seen by the ophthalmoscope.

November 4th: Lens again in anterior chamber. Patient kept on right side. Lens 5 mm. in diameter with coloboma below. Under a local anes-

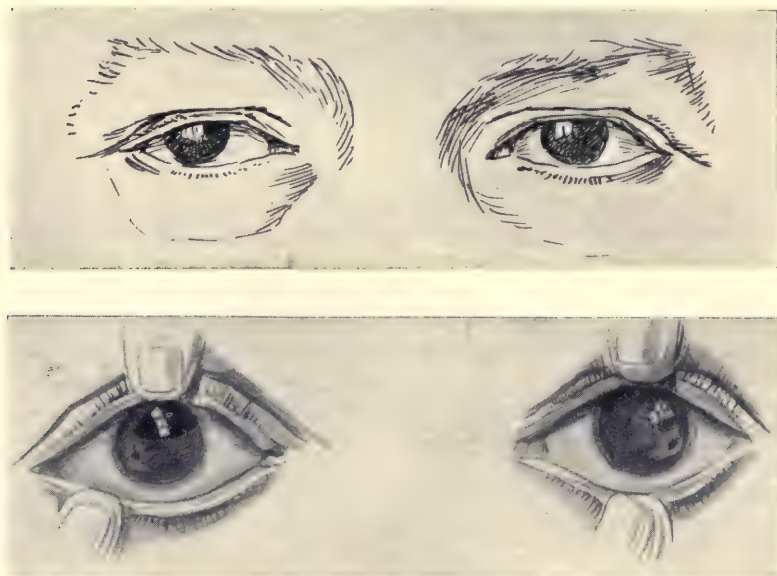
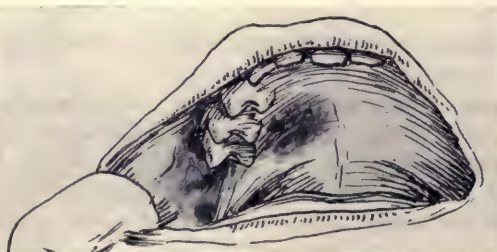
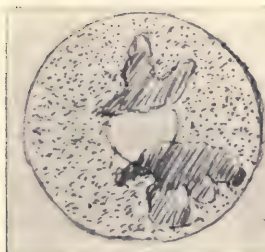


Fig. 1.—Bilateral symmetric congenital corectopia after operation on right eye.

thetic a section of the cornea was made up and out. Lens spooned out. In the process the capsule was ruptured partially. Whole drawn out by forceps; atropin instilled.

November 10th: Some swelling of lids, slight injection, and chemosis of the conjunctiva. Keratitis striata. Fair anterior chamber. Coloboma larger than before extraction of lens.

December 3d: Right palpebral fissure smaller than left. Slight redness of skin of lids. Merest trace of limbal injection. Wound perfectly healed. Cornea bright. Fair anterior chamber. The new pupil is absolutely black. The original pupil is almost closed by sphincter and shows as black, irregular



Appearance of iris like fine brown velvet, with two patches of brown jelly lying on surface.

Palate

Fig. 2.—Melanosis of right eye, scalp, cheek, and palate.

line only. V.R.E. + 9 D. sph. \ominus + 2 D. cyl. ax. $15^\circ = 6/36$; + 16 D. sph. \ominus + 2 D. cyl. ax. $15^\circ = J. 8$.

Vitreous clear. Disc is somewhat blurred at margin. Staphyloma posterior disc $\frac{1}{2}$ diameter in width. Superior temporal retinal veins tortuous. No other fundal lesion. The left field shows slight contraction, but the right is normal. There is no scotoma. Tension normal.

MELANOSIS OF THE EYE, SKIN OF RIGHT SIDE OF HEAD, AND RIGHT PALATE

Ethel Mary R., female, aged twelve.

History.—One sister aged ten; one sister aged seven, all healthy. One half brother, seven months, quite well.

No history of any abnormal pigmentation in two maternal uncles, eight maternal aunts, three paternal uncles, two paternal aunts. Has numerous cousins, all well.

Patient had slight scarlet fever and varicella.

There is an extensive patch of pigmentation of the skin of the right side of the head, affecting the upper part of the cheek, the temple, and the skin of the scalp, reaching nearly to the vertex. The density of the pigmentation is not uniform, and it resembles the color of a blue-black ink-splash. The edges of the area are irregular and not sharply defined. The skin is not swollen and does not fade on pressure. There is no nevoid condition.

A similar pigmentation is found on the right palate, with indistinct margins, and a less deeply pigmented area is seen in the mucous membrane of the right cheek. The patch on side of cheek and temple fades a little when weather is fine, and goes darker when wet. The pigmentation was present at birth and has not changed. No other abnormal pigmentation is discernible. The right globe presents the most striking feature of the case. It is somewhat enlarged and divergent to the extent of some 15 degrees. The palpebral fissure is a trifle wider than the left, exposing the sclera below for about 1 mm. The conjunctiva itself is not affected. The ocular portion is free from any abnormal adhesion to the episcleral tissue. The latter presents extensive areas of brown-black pigmentation. Two of the better defined areas are distinctly swollen to the extent of 1 mm., viz., the large one below the cornea and a smaller one above. The cornea shows in a small central area fine superficial pitting, but it is otherwise clear. The anterior chamber is rather shallow. The surface of the iris is a stippled dark brown, as seen in the brown eyes of dark races. There are two areas of brownish, gelatinous material, apparently lying on it, both of which extend slightly over the pupillary margin. The anterior capsule and cortex are clear. Some bluey opacity in the nucleus of the lens is easily seen. No red reflection is obtained by the ophthalmoscope. V. R. E.: No l. p. No lighting of pupil by transillumination. T + 1.

The left eye is normal in every respect and its field good.

The thyroid is large; some thrill felt on pressure on gland. Impairment of resonance with some collapse of the lung at the right apex, but no evidence of active disease.

There is a deep-seated mass in the right hypochondrium, which is moderately tender on pressure (glands?).

Vascular and nervous systems normal.

DISCUSSION

MR. E. TREACHER COLLINS (London, England): I have been much interested in the subject of melanosis of the eye and the appearance of the iris in such cases. Some years ago the late Dr. George Coates showed a case of this description in which on high magnification of the surface of the iris there was a very peculiar appearance. It presented a number of little nodular elevations very much like the elevations on a golf ball. At the same meeting I was able to show a case of melanosis in which the surface of the iris presented a shaggy appearance which might be compared to that of an India rubber sponge. Then about two years ago Dr. Sterling, of Atlanta, Ga., had a case of melanosis of the iris in which he had occasion to remove a portion of it, and he kindly sent me the fragment of iris to examine microscopically: It showed that on the anterior surface there was a marked hyperplasia of the endothelium of the iris, and this accounted for the irregularity of pigmentation of the surface.

I would like to ask Mr. Clegg whether he had examined the surface of the iris in his case under high magnification, and if so whether he found any such appearance.

DR. E. E. BLAAUW (Buffalo, N. Y.): Mr. Clegg has mentioned that the extracted lens in the one eye had a 5-millimeter dimension. This points to Vogt's conception of "embryonal nucleus" being a definite stage in the lens development, a stage which can be followed through man's complete life.

DR. EDWARD JACKSON (Denver, Col.): The anomaly of pigmentation, apparently general and not strictly local, certainly on one side, leads us back to the thought of the radical relation which pigmentation seems to have with the vital processes of development, the general biologic processes. It suggested to my mind, on reading Mr. Clegg's case, that probably in that eye would be found some other radical departures which would account for the complete blindness. The suggestion made that possibly it is going on to tumor development, sarcoma of the eye—that is a possibility from the doubtful rise of tension—gives an obvious explanation; but even such a development may be associated with the radical connection between pigmentation and the development of organs that normally contain pigment.

MR. J. GRAY CLEGG (closing): In reply to Mr. Collins, I would say that the iris under strong magnification presented the appearance of very fine dark brown velvet, and lying on the iris near the pupillary margins were two gelatinous, flattened, irregular membranes, extending on to the pupil itself.

LOS MERCURIALES, EL SALVARSAN Y SUS DERIVADOS EN EL TRATAMIENTO DE LAS FORMAS GRAVES DE CONJUNTIVITIS Y QUERATITIS ECZEMATOSAS

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La conjuntivitis eczematosa, conocida también con los nombres de flictenular, impetiginosa, estrumosa, vascular, oftalmía linfática, y recientemente con el nombre de conjuntivitis tuberculosa, es sin duda una de las enfermedades que se presentan con más frecuencia al oftalmólogo. La gran mayoría de los casos corresponden a formas benignas, que ceden con relativa facilidad al tratamiento clásico; pero también, con bastante frecuencia, se presentan formas graves cuya evolución crónica, su tenaz reincidencia y la rebeldía a todo tratamiento, constituyen un verdadero problema para el médico tratante. En cuanto a las consecuencias de estas formas graves son dignas de tomarse en consideración y de temerse por los daños, a veces irreparables, que producen en la cornea, consistentes en panus, nefelios, infiltraciones, esclerosis y leucomas, con o sin enclavamiento del iris. En efecto, la participación de la cornea en las formas graves es constante y de aquí que los síntomas sean tan molestos y tan alarmantes y las consecuencias tan severas.

Esta enfermedad, peculiar a los niños de 3 á 15 años, encierra hasta la fecha el misterio más completo sobre su etiología. En verdad pocos puntos hay en Medicina tan oscuros como el presente, en el que la tenacidad y laboriosidad de los investigadores ha fracasado. La literatura sobre el tema es enorme, y por lo tanto sólo me limitaré a hacer una breve exposición de las ideas y de los hechos más culminantes, así como de las opiniones más autorizadas.

Las causas hasta ahora señaladas pueden clasificarse de la siguiente manera: A. Causas Constitucionales; B. Causas tóxicas; C. Causas microbianas; D. Causas parasitarias, y E. Causas neurotróficas.

(A) Desde hace muchos años se ha atribuido al terreno un papel preponderante en esta enfermedad, de aquí los nombres de conjuntivitis escrofulosa, estrumosa, oftalmía linfática, que ha recibido desde antaño; en efecto, desde entonces se había visto que los portadores de ella eran en su mayoría individuos escrofulosos, cuya circunstancia hizo que se llegara a considerar la presencia de esta conjuntivitis como el signo más eficaz y constante del temperamento linfático.

La alimentación deficiente, las malas condiciones higiénicas, la dentición y la convalecencia de las enfermedades infecciosas agudas, han sido otras tantas causas invocadas, cuya acción se refleja directamente sobre el terreno.

(B) Burns, Colombo, etc., han señalado entre las causas de este grupo la autointoxicación por retención de materias estercoreales.¹ Turner, en un estudio sobre la etiología de esta enfermedad, ha señalado como causa la toxemia, debida principalmente a trastornos dependientes del aparato respiratorio: adenoides, hipertrofias amigdalinas y de los cornetes nasales; así como infecciones de la nariz y de los senos anexos, cuyos trastornos, a su vez, los subordina a la autointoxicación gastro intestinal.²

(C) Contra la opinión casi unánime de los oftalmólogos, Leber y Sattler, Gifford y otros investigadores, han atribuido esta enfermedad al estafilococo, que han logrado aislar de la secreción conjuntival de algunos enfermos; mas semejantes ideas no han sido admitidas y la interpretación general que se ha dado a dichos hallazgos, es considerar la presencia de tales gérmenes como ocasional y no como causal. En efecto, contra la posibilidad de una causa microbiana, están las experiencias de Terrien, quien ha inyectado en la cámara anterior de monos, conejos y cueros, diversos productos obtenidos de las flictenas, sin haber logrado ninguna inoculación.³ Por otra parte, Saemisch, quien designó la enfermedad con el nombre de conjuntivitis flictenular, llegó también, por una serie de investigaciones, a la conclusión de su no inoculabilidad. Los trabajos en este sentido han sido numerosos y todos convergen hacia la misma conclusión.

Axenfeld, Dor, Weckers y algunos otros oftalmólogos, atribuyen la enfermedad a la acción debilitada de las toxinas tuberculosas, cuyo origen sería habitualmente algún foco tuberculoso distante del ojo, pulmonar, oseó, ganglionar, etc., Wolff ha lanzado la teoría de que las flictenas no serían sino una manifestación anafiláctica de la conjuntiva, consistente en una hipersensibilidad de ella a las toxinas tuberculosas

atenuadas, procedentes habitualmente de algun foco ganglionar del cuello y supone, que el ojo mismo, haya sufrido con anterioridad algún padecimiento bacilar, del cual ha sanado por los procesos naturales.⁴

Gibson en 92 casos de conjuntivitis flietenular ha encontrado 90 con von Pirquet positivo y ha logrado producir flietenas en conejos tuberculosos mediante la instilación de tuberculina en los fondos de saco conjuntivales.⁵ Weckers en 55 casos encontró 51 con reacción positiva a la misma prueba.⁶ Wesseley señala un 95 por %.⁷ Köllner ha estudiado 140 casos de la enfermedad con relación a diversas manifestaciones tuberculosas de que eran portadores los enfermos, y encontró una relación entre la evolución de éstas y la enfermedad ocular.⁸ En México Fernandez MacGregor señala un 98% de von Pirquet positivo.⁹ Pero en contra de estos datos positivos hay que señalar otros diametralmente opuestos. Asi, el Dr. Pacheco Luna de Guatemala ha declarado que, tanto los exámenes radiológicos como las pruebas de von Pirquet, han sido constantemente negativas.¹⁰ Burnett ha encontrado una proporción muy inferior a las señaladas, y niega que haya una relación de causa a efecto entre la enfermedad y la tuberculosis.¹¹ Igualmente opina Morax a este respecto.¹²

Por mi parte puedo añadir que en compañía del Dr. Daniel M. Velez he practicado la prueba de Calmette en varios niños de la Casa de Expósitos de México y pude observar, contra lo que me espera, que aquellos que habían padecido poco antes de conjuntivitis flietenular, dieron constantemente una reacción negativa.¹³

(D) Chevalereau, Axenfeld, Pacheco Luna, y algunos más, han indicado la coincidencia de esta enfermedad con la existencia de otiriasis en los niños portadores de la enfermedad, y atribuyen la causa a la acción tóxica de la saliva del parásito que es introducida en el momento de la picadura.¹⁴

Además de los piojos han sido considerados como causa de esta conjuntivitis, los oxiuros, entre otros autores por el Dr. Andrade de Brasil, quien ha logrado producir experimentalmente la enfermedad en el cachorro, por instilación en la conjuntiva, del líquido perientérico de los expresados vermes.¹⁵

(E) Se ha pensado que esta enfermedad tiene alguna analogía con el herpes zona, lo que ha recibido confirmación por el hecho real de que las partes preferentemente afectadas en la cornea, corresponden frecuentemente a las terminaciones nerviosas, lo que justifica en cierto modo la sintomatalogía tan aparatosa de algunos casos.

De lo anteriormente expresado se deduce que hasta la fecha no hay

en definitiva una causa a que atribuir la enfermedad, cuya etiología queda aun por establecerse; pero pueden asentarse tres hechos perfectamente probados y generalmente admitidos:

1. La enfermedad se encuentra habitualmente en niños estrumosos; aunque puede presentarse en algunos de constitución aparentemente sana.

2. Tiene marcada relación con las lesiones impetiginosas de la nariz, de la cara y aun de otras regiones del cuerpo; por lo que le viene los nombres de eczematoso, impetiginoso y pustuloso.

3. Que es amicrobiana y por lo tanto no inoculable.

Por mi parte puedo señalar un hecho más que hasta la fecha no ha merecido reparo, o bien que ha sido negado y es su frecuencia en niños heredo sifilíticos comprobados.

En efecto, no alcanzo a comprender, como siendo relativamente grande el número de niños afectados de esta enfermedad, que presentan al mismo tiempo signos o estigmas evidentes y aun culminantes de dicha infección, no haya llamado la atención de los investigadores. Sus pesquisas no se han orientado en este sentido y, en cambio, si han sido de preferencia sobre la tuberculosis, siendo esta infección más discreta y más difícil de comprobar por tener que valerse, casi de una manera exclusiva, de medios de laboratorio.

No sólo esta enfermedad es frecuente en los niños heredo sifilíticos, sino que la mayor parte de las veces que la he visto en los adultos, se trataba de sujetos portadores de sífilis. Por otra parte, no es extraño observar formas asociadas de esta enfermedad con alguna otra manifestación netamente específica, como la de una queratitis intersticial, una escleritis profunda, una iridociclitis, etc., datos sobre los cuales tampoco se ha hecho la mención debida y apenas si Wecker y Landolt, mencionan en su obra, la asociación de la conjuntivitis eczematoso con las epiescleritis.¹⁶

Esto no quiere decir que señale de una manera categórica a la sífilis como causa de la conjuntivitis eczematoso; pues carezco hasta estos momentos de las pruebas y los fundamentos necesarios; pero como quiera que he sido uno de los primeros en mencionar esta causa volveré a tratar del asunto al final de este trabajo.

TRATAMIENTO POR LOS MERCURIALES

Todos los autores están conformes en que los mercuriales en forma de colirios son específicos contra esta enfermedad, y desde hace mucho tiempo se vienen prescribiendo. El calomel y el óxido amarillo

de mercurio recientemente precipitado, han sido los dos medicamentos más puestos en boga; también el bicloruro de mercurio, el cinabrio y el óxido rojo, han sido usados, aunque menos extensamente que los anteriores.

Con relación a la manera como obran estas substancias, nada se sabe; algunos creen que es debido a la descomposición lenta que sufren en presencia de las lágrimas, y otros les han atribuido una acción puramente mecánica, que experiencias posteriores han venido a dementir.

Algunos autores han usado las sales de plata, como el argirol y el protargol, pero su acción es indudablemente inferior a la de los mercuriales.

El Dr. Menacho recomienda el uso de colirios con tanino; pero personalmente me he convencido de su inferioridad con el calomel.

Por último Colleman, Wolff, etc., aplican los rayos X asegurando buenos resultados. Este último autor no sólo ha logrado mejoría en las lesiones oculares, sino que aun en las de los ganglios del cuello, y usa para la aplicación ocular 4 unidades H.¹⁷

Por mi parte puedo decir que uso un colirio de calomel a 10 % en suspensión en vaselina líquida, del que estoy muy satisfecho.

Como tratamiento general los clásicos aconsejan el aseo, la buena alimentación, buena ventilación y toda clase de preceptos higiénicos encaminados a mejorar la salud del paciente. Además indican alguna medicación tónica o antiescrofulosa, como los glicerofosfatos, el aceite de hígado de bacalao, el iodo, etc.

Hasta la fecha no se ha mencionado un tratamiento general, que obre de una manera enérgica y eficaz sobre la evolución del padecimiento ocular, de que me vengo ocupando, como obraría el tratamiento específico en una iritis o en una coroiditis de origen sifilítico. En las querato conjuntivitis flictenulares de forma grave, tanto el tratamiento local como el general hasta ahora aconsejados, son ineficaces, pues hay casos en que la enfermedad se prolonga indefinidamente y que si curan, es espontáneamente, cuando los medios naturales tienden a hacerlos regresar. El resultado de esta evolución tiene sobre la corena las fatales consecuencias que antes he señalado, y no siendo aun bastante, queda el enfermo constantemente propenso a recaer en su enfermedad.

Desde hace seis años pude accidentalmente darme cuenta de que el mercurio introducido al interior del organismo se mostraba eficaz

contra la enfermedad, determinando una acción decididamente favorable sobre la evolución de los casos más severos.

Hasta ahora el tratamiento general por el mercurio en este padecimiento no ha sido mencionado, con excepción de Bruns, quien recientemente e independientemente de mí, lo ha manifestado.¹⁸

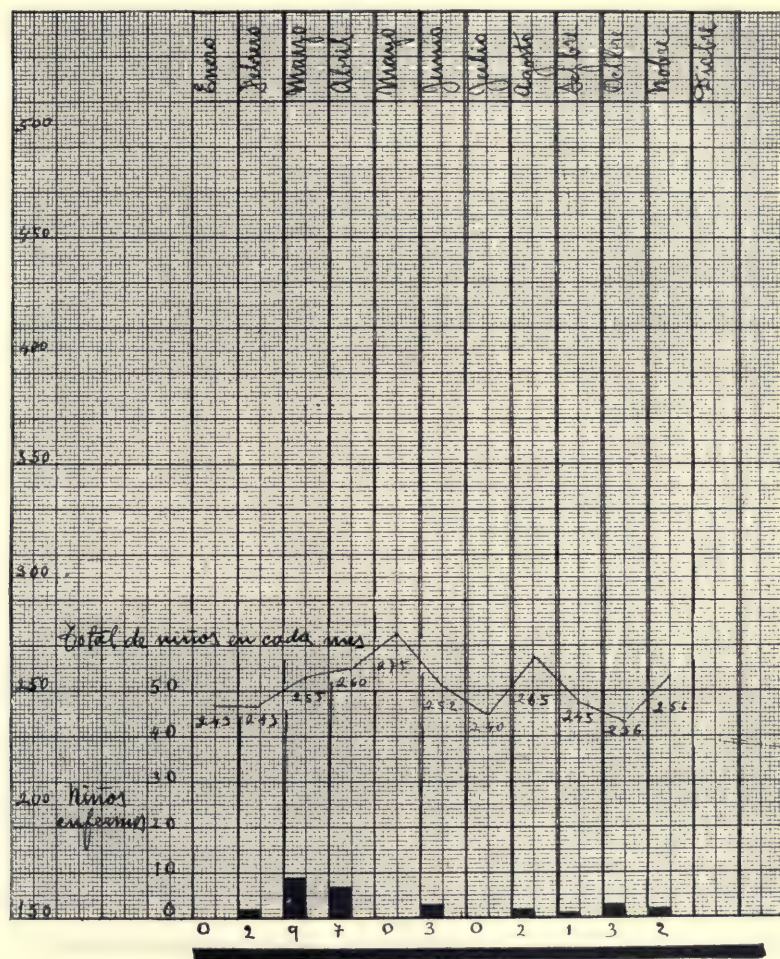
Posteriormente, como se verá en el curso de este trabajo, he usado con éxito aun mayor los derivados del salvarsan, a cuya circunstancias debo el honor de trazar estas líneas.

Me creo en la obligación de decir como llegué al conocimiento de estos hechos, y por lo tanto transcribo la siguiente nota clínica que fué para mí de grandes enseñanzas:

El año de 1915, atendía de queratitis parenquimatosa a un niño, en quien la sífilis estaba fuera de toda duda, a juzgar por los estigmas de que era portado. Al mismo tiempo una hermanita, cuatro años mayor que él, había sido internada en un sanatorio de esta ciudad, para ser tratada de múltiples y enormes ganglios infartados del cuello. Se le practicó la extirpación de los ganglios estrumosos; pero la operación fué seguida del fracaso más completo: algunos ganglios sanos que quedaron, a su vez se infartaron; el cuello en general estaba invadido de una enorme empastamiento y presentaba una rubicundez violacea; las heridas operatorias se habían retrasado en cicatrizar y secretaban una serosidad espesa que se secaba formando costras; la deglución era muy difícil; los movimientos del cuello eran casi imposibles y la enfermita apenas si dormitaba reclinada entre almohadones. La desnutrición cada día se acentuaba y el caso llegó a ser tan alarmante que se abrigaron los temores de un desenlace fatal.

En los días en que se iniciaba esta gravedad apreciaron en un ojo varias flictenas, le hice una visita a la paciente y receté un colirio con calomel; pero la enfermedad ocular se empeoraba al mismo tiempo que el estado general. Por último la enfermita fué trasladada a su domicilio con la idea de que fuera a morir en el seno de su familia. Le practiqué una segunda visita y entonces, en un momento de dolor en que abrió demesuradamente su boca, pude observar que presentaba unos dientes de Hutchinson enteramente típicos y una vóveda paltina ojival, cuyos datos, unidos a la queratitis intersticial del niño, y algunos otros que pude recoger por el interrogatorio, me hicieron recordar la expresión inmortal de Fournier: "La escrófula es un escrofulato de verole." En el acto concebí la esperanza de que un tratamiento específico mejoraría a aquella niña tan gravemente enferma, y ese mismo día le apliqué una inyección intravenosa de cianur de mercurio de

0.01. Después de ocho inyecciones, el resultado era verdaderamente milagroso: Las heridas operatorias habían cicatrizado; el empasta-

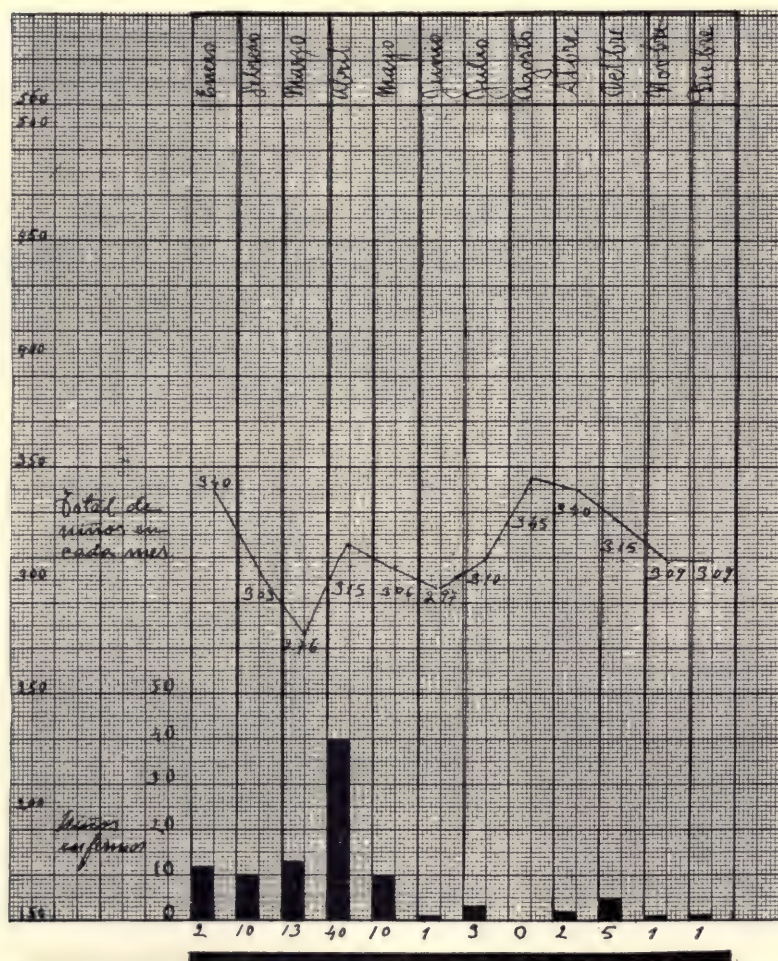


Año de 1921

Casa de Niños Expósitos.—Datos relativos al número de niños enfermos de conj. flictenular y a la existencia mensual

miento del cuello había desaparecido; la enferma dormía y comía, con lo que recobraba visiblemente fuerzas y ánimo. En cuanto a la lesión ocular, que se había declarado tan revelde al tratamiento local,

evolucionó de una manera tanto o más admirable que el estado general de la enferma.

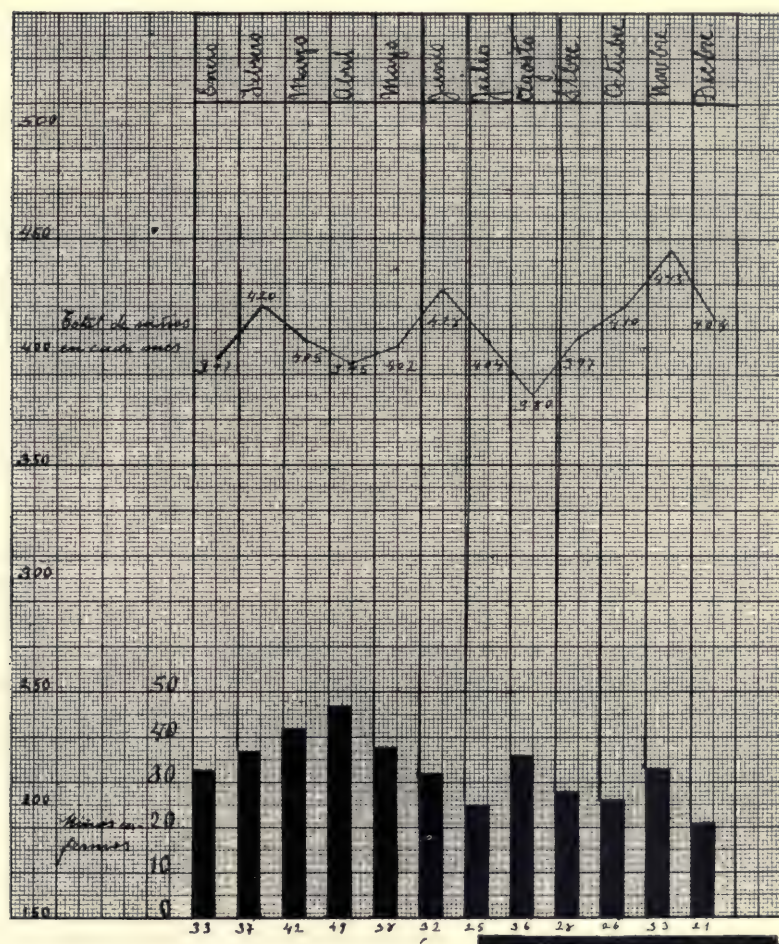


Año de 1920

Casa de Niños Expósitos.—Datos relativos al número de niños enfermos de conj. flictenular y a la existencia mensual

Poco tiempo después se me presentó otro caso de conjuntivitis flictenular de mediana intensidad, en una niña con datos negativos de heredo sífilis, pero muy revelde al tratamiento local, y vivas como

tenía las enseñanzas del caso anterior, no vacilé en inyectar en la región glútea una solución de 0.01 de cianuro de mercurio. Al día



Año de 1919

Casa de Niños Expósitos.—Datos relativos al número de niños enfermos de conjuntivular y a la existencia mensual

siguiente había una notable mejoría, obteniéndose una curación completa mediante dos inyecciones más.

Desde entonces comencé a usar el mercurio como tratamiento

general en los casos de conjuntivitis eczematosa, obteniendo cada vez resultados altamente satisfactorios. Pero como en la mayor parte de los casos se trata de niños, repugna hacerlos sufrir por las inyecciones intramusculares de sales hidrargíricas, o bien por que se dificulta sobre manera la inyección intravenosa, me he decidido por las fricciones de unguento mercurial, con no menos buenos resultados.

Este tratamiento es el que uso en la clientela de hospital, en el dispensario del Servicio Higiénico Escolar y en la Casa de Niños Expósitos.

Acompaño tres gráficas con datos tomados de la Casa de Niños Expósitos, en las que están expresos la existencia total de niños en cada mes y el número de enfermos de conjuntivitis eczematosa. La primera, que corresponde al año de 1919, indica un número muy alto de enfermos, coincidiendo con la ausencia del tratamiento mercurial. La del año de 1920, época en que me hice cargo del Servicio, arroja ya una disminución apreciable del número de casos, debido a que los pacientes comenzaron a ser tratados por las fricciones mercuriales. La gráfica del presente año, comparada con las anteriores, señala una desproporción enorme, aun teniendo en cuenta que el número de niños ha sido menor que en los años anteriores.

El resultado del tratamiento mercurial sobre el padecimiento de que me vengo ocupando, no puede ser más ostensible. La disminución gradual que se ha conseguido, la atribuyo a que la mayor parte de estos niños tienen frecuentemente padecimientos nasales impetiginosos, que también mejoran rápidamente con el tratamiento mercurial, y algunos son francamente heredo específicos, lo que me ha hecho insistir con relativa frecuencia en él; y no sólo se ha logrado la reducción señalada en el número de casos, sino que cuando estos se han llegado a presentar, es de una manera esporádica y han sido de una benignidad notable. Como confirmación a este dato puedo manifestar que en el curso del presente año, los casos graves no han excedido de dos.

TRATAMIENTO POR EL ARSENOBENZOL Y SUS DERIVADOS

La acción terapéutica enérgica del mercurio en el tratamiento de los casos graves, así como la frecuencia de algunos estigmas, o por lo menos indicios de sífilis en los pacientes, me hicieron probar otros agentes antisifilíticos y no vacilé en inyectar dosis de los derivados del arsenobenzol, siendo el resultado el más alhagador y sorprendente que se pudiera esperar, pues su acción supera notablemente a la ya grande abtenida por los mercuriales.

Los casos clínicos que a continuación relato, darán una idea clara de esta terapéutica.

A. G. de L. Niña de seis años. Constitución debil y muy delicada. Su padre murió hace cuatro años de tuberculosis pulmonar. No hay más antecedentes de tuberculosis en la familia. A la edad de cuatro años, la pequeña tuvo un padecimiento gastro-intestinal muy revelde. No presenta estigmas ni antecedentes de heredo sífilis. No presenta ganglios infartados, ni cicatrices en el cuello. Hace un año cuatro meses viene padeciendo de una querato conjuntivitis eczematosa en ambos ojos, con pequeñas intermitencias y más acentuada en el ojo derecho. El examen reveló en este ojo multiples flictenas en el limbo y algunas en la cornea y ésta cubierta de panus. La fotofobia era intensa y los párpados presentaban escoriaciones en el ángulo externo. El ojo izquierdo presentaba algunas pequeñas flictenas en el limbo y algunos nefelios, que testificaban ataques anteriores de la enfermedad y la participacion de la cornea en ellos. La prueba de von Pirquet fué negativa. Tratamiento: colirio de calomel y aplicación de una dosis de 0.10 de Musebarsenol Corbiere, lográndose días después una mejoría muy apreciable. Seis días más tarde apliqué una dosis de 0.15 y el alivio se acentuó más. Al cabo de tres días de la tercera inyección, que tambien fué de 0.15, ya no se veían las flictenas; el panus había desaparecido a la simple vista y sólo la lente revelaba toda—via algunos vasos; lo único que aun persistía, pero muy mejoradas, eran las escoriaciones de los párpados. Fueron aplicadas dos inyecciones más de la misma dosis y se obtuvo una curación completa, mejorando al mismo tiempo el estado general de la niña.

C. N. de 15 años. Constitución debil. Presenta antecedentes hereditarios que suponen la sífilis. Nulos respecto a la tuberculosis y el von Pirquet negativo. No presenta infartos ganglionares del cuello. No hay estigmas de heredo sífilis, pero la reacción de Wassermann es dos veces más. Desde su infancia viene padeciendo ataques de conjuntivitis eczematosa en ambos ojos y de una manera más insistente en el ojo izquierdo, en los dos últimos años. El examen reveló numerosas flictenas conjuntivales y corneales en ese ojo; una úlcera en el limbo, como a las 9 h/; panus y algunas infiltraciones en la cornea. La fotofobia era muy intensa. La visión estaba muy disminuida, al grado de no ver si no el bulto de la mano.

Tratamiento: colirio de calomel, atropina y vendaje. Previo reconocimiento de orina, se le aplicó una dosis de 0.15 de neosalvarsan por la via endovenosa, desapareciendo días más tarde, la fotofobia y pudiéndose dejar el ojo al descubierto por haber cicatrizado la úlcera. El medicamento se fué inyectando en serie cada seis días hasta llegar a la dosis de 0.45, y se obtuvo después de cuatro inyecciones un alivio tan completo, que la agudeza visual llegó; a 0.3. En la cornea persistían, sin embargo, nefelios y algunas infiltraciones gruesas, muy blancas y vascularizadas.

J. S. Sexo femenino de 19 años. Constitución muy debil y delicada. Ha

tenido quince hermanos, de los cuales ocho han muerto, tres antes de dos meses de nacidos y los cinco restantes, de pocos años. Uno de los que viven es loco. Otro es marcadamente escrofuloso. La madre padece de epilepsia. El padre vive y es aparentemente sano. La paciente no presenta infartos ganglionares del cuello ni estigmas de heredo sífilis y la reacción de Wassermann fué negativa. Desde pequeña tuvo varios ataques de conjuntivitis eczematosa, permaneciendo algunos años libre de ellos; pero desde hace seis años ha vuelto a tener brotes de ella. Hace seis meses se queja de tos. Se ha acentuado su enflaquecimiento y a veces por las tardes, tiene elevaciones de temperatura y sudores profusos. El padecimiento que la ha traído a consulta data de tres meses, encontrándose al examen del ojo izquierdo una conjuntivitis flictenular de forma grave, con panus, flictenas en la cornea y dos úlceras pequeñas de bordes netos y redondos, como hechas con sacabocado y con insignificante infiltración de ellos y del fondo. La fotofobia era tan intensa, que la enferma ocultaba su cabeza entre el colchón y las almohadas, y había además una intensa cefalea. El ojo derecho acusaba en la cornea huellas de varios ataques del padecimiento. El examen general de la enferma hizo presumir una tuberculosis pulmonar incipiente. El von Pirquet fué debilmente positivo.

No obstante los datos anteriores se comenzó a inyectar neosalvarsan por la vía endovenosa, obteniéndose una curación tan rápida que a los tres días de la segunda inyección, las úlceras estaban espejeantes, el panus muy adelgazado y la fotofobia y la cefalea habían desaparecido. Después de la tercera inyección fué necesario suspender el tratamiento, en vista de la dificultad para puncionar las venas a la paciente y este descanso determinó la reaparición de algunas flictenas y un pequeño engrosamiento del panus. En vista de lo cual le fué aplicada una dosis de 0.30 en la yugular, haciéndose sentir más tarde los efectos benéficos. La enferma curó después de un tratamiento de seis inyecciones. El estado general mejoró notablemente y desaparecieron muchos de los síntomas que hacían presumir la tuberculosis pulmonar.

DISCUSIÓN

Es innegable que la mayor parte de las conclusiones relativas a la etiología de este padecimiento, convergen hacia la hipótesis de su origen tuberculoso, y digo hipótesis porque hasta la fecha no se ha señalado la presencia del bacilo de Koch en las flictenas, ni tampoco de una manera constante en el organismo de los enfermos. Esta uniformidad de criterio contrasta desde luego con la acción casi nula de toda terapéutica antituberculosa, incluyendo las tuberculinas y con los dos hechos fundamentales que antes he señalado: su frecuencia en pacientes sífilíticos y su curación pronta por los mercuriales administrados ocal y generalmente, así como por los derivados del salvarsan.

El problema adquiere una complicación mayor por el hecho frecuentemente observado y citado por algunos autores alemanes, entre ellos Zehener, quienes han señalado la excesiva coincidencia de la sífilis con la tuberculosis, y se suponen que la primera de estas enfermedades prepara favorablemente el terreno debilitándolo, para que pueda desarrollarse con más facilidad la segunda. Además han logrado tratar y curar por un tratamiento antisifilítico algunos trastornos que indudablemente no eran de origen tuberculoso.¹⁹

De los datos que dejo consignados, así como de otros muchos que no hubiera sido posible relatar, se desprende que tanto la sífilis como la tuberculosis pueden estar presentes en el enfermo, solos o en concurso; pero de todas maneras el problema acerca de la etiología del padecimiento queda en pié, pues como lo he indicado, las valiosas pesquisas de aptos y respetables investigadores, no han llegado a demostrar en él la naturaleza tuberculosa, ni menos por los datos que llevo consignados debe deducirse un origen sifilítico.

Sin embargo, la hipótesis de que en un gran número de casos pueda atribuirse a la sífilis el padecimiento, no es tan despreciable. En efecto, desde Iwanoff quien fué el primero en estudiar la anatemia patológica de las flictenas, se ha comprobado que estas lesiones corresponden a inflamaciones del tipo nodular en su fase inicial, lo que ha hecho mirar a los partidarios del origen tuberculoso un pequeño tubérculo en la flictena; pero precisamente esto sería lo que daría algún fundamento al origen sifilítico de la lesión. Realmente, el microscopio difícilmente podría decir si una granulación pequeña y reciente, supongamos de las meninges, sería sifilítica o tuberculosa, pues ambas tiene exactamente la misma forma de infiltración linfocitaria, formación de celdillas gigantes, etc., y sólo la distinción es posible cuando el examen se lleva sobre lesiones más avanzadas, en las que ya es posible observar las zonas necróticas, disminución de la irrigación sanguínea, etc., en el tubérculo; o bien el tejido de neoformación, esclerosis y demás caracteres, que identifican las lesiones sifilíticas.

Bien pudiera decirse que esta hipótesis va tomando forma, pues en los casos de conjuntivitis flictenular de duración muy prolongada y en la queratitis fascicular, algunos autores, entre ellos Gruber, Hertel, Augstein, Yamaguchi, Baas, etc., han señalado en la capa de Bowman y aun debajo de ella, invadiendo el tejido propio de la cornea, algunas esclerosis y producciones de tejido de nueva formación. De estos autores el último mencionado, no sólo ha demostrado estas lesiones,

sino que eran frecuentes en pacientes sifilíticos y pudo además demostrar en los ojos exminados, algunas lesiones de coroiditis sifilítica.²⁰

Estos datos, unidos a la no inoculabilidad del padecimiento; a la ausencia de microorganismos patógenos en las lesiones, por más empeño que ha habido en descubiertos, y a la acción curativa que tienen los agentes antisifilíticos, robustecen la hipótesis del origen sifilítico de la conjuntivitis flictenular, si nó en todos los casos, por lo menos en una gran parte.

Carezco por completo de la autoridad y de los fundamentos necesarios para asentar categóricamente esta aseveración; pero quedaría altamente honrado y satisfecho si con lo que he dicho hasta ahora abriera nuevas vias de investigacion, fueran o no coronadas por el éxito.

De todas maneras están patentes las conclusiones que hacen presumir el origen tuberculoso de la enfermedad y no creo que sea tan facil destruirlas.

En cuanto a la acción curativa de los mercuriales, no sé realmente a qué atribuirla, sobre todo en ausencia de sífilis en el enfermo. Queda la posibilidad de considerarles propiedades terapéuticas distintas de las señaladas.

Mucho se ha dicho de su acción resolutive; pero ésta cada día pierde terreno por encerrar una idea vaga, muy poco satisfactoria, si no es que muchas veces erronea.²¹

En cuanto a los derivados del salvarsan hay una gran tendencia a emplearlos en diversidad de padecimientos que no son sifilíticos ni hematoparasitarios, tales como la piorrea, el reumatismo, la oftalmía simpática (Morax²²), la gangrena pulmonar (Svolin y Sjöblom²³), etc. Pero la mayoría de los autores que emplean dichos derivados arsenicales en los padecimientos no sifilíticos, no indican la manera como obran.

Tratándose de enfermos sifilíticos tampoco es facil darse cuenta exacta de la manera como obran los agentes terapéuticos, mercurio y salvarsan, pues no habiéndose demostrado la naturaleza sifilítica del padecimiento, no se puede atribuir a esta circunstancia su curación.

En los casos en que la sífilis y la tuberculosis concurren en el paciente, la explicación se hace más incomprensible y se podría sentar la hipótesis de que estos casos serían más favorecidos que aquellos en que solo existe la tuberculosis, debido a que los agentes específicos obrarían de una manera más segura, robusteciendo al organismo; lo cual se podría lograr por un camino indirecto, o sea estimulando al

organismo por una medicación tónica, mejorando la alimentación y las condiciones higiénicas.

CONCLUSIONES

1. Los casos graves de conjuntivitis eczematosa constituyen aun un problema muy serio para el oftalmólogo y una amenaza para la vista de los niños quienes son los que habitualmente padecen esta enfermedad.

2. La etiología del padecimiento por ahora es completamente desconocida. Generalmente se atribuye a las toxinas tuberculosas; pero no es ésta la única causa.

3. La sífilis es frecuente en los niños afectados de la enfermedad, hecho que casi no ha sido mencionado; pero no hay fundamentos suficientes para atribuir el padecimiento ocular a dicha enfermedad.

4. El mercurio y los derivados del salvarsan, tienen una acción marcadamente específica contra el padecimiento.

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THE EFFECTS OF DIRECT INSTILLATION OF NOV- ARSENOBILLON IN THE CONJUNCTIVAL SAC IN RESISTANT CASES OF CONGENITAL SYPHILITIC INTERSTITIAL KERATITIS

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In any extensive series of cases of interstitial keratitis of syphilitic origin one comes across a number which are very resistant to ordinary mercurial treatment. At the Royal Eye Hospital in Manchester these cases, and in fact most of the ordinary type, are submitted to treatment by means of salvarsan or its derivatives, novarsenobillon, neosalvarsan, or neokharsivan. Of these I have been able to distinguish three types:

1. The type which rapidly improves under combined mercurial and arsenical treatment.
2. The type which rapidly improves but subsequently relapses under this treatment.
3. The type which, even after prolonged administration of mercury, iodids and salvarsan derivatives fails to improve in so far as the infiltration of the cornea is concerned. Having noted that a definite ground-glass or hazy appearance of the cornea persisted even after twelve to fifteen injections of novarsenobillon or neokharsivan, it

occurred to me that the direct instillation of concentrated solutions of the novarsenobillon might be of use. In view, however, of the irritant action of this drug on the subcutaneous tissues, I discussed the matter with Dr. J. Gray Clegg, of the Manchester Royal Eye Hospital, and we decided that it was advisable to ascertain the effects on the eyes of animals before using it clinically on human beings. A 1% solution of N. A. B. in distilled water was prepared and instilled into the conjunctival sacs of the eyes of rabbits. The results were examined by Dr. Clegg and myself, and he decided that there appeared to be nothing to contraindicate its use in children. Accordingly a number of patients were submitted to me by him for this method of local treatment.

Subsequently other members of the honorary staff of the Manchester Royal Eye Hospital sent cases for similar applications. All the above-mentioned types have been dealt with; namely, the early type, the relapsing type, and the resistant type. In the two former types general anti-syphilitic treatment was in its earlier stage and it is difficult to appraise in a scientific manner the exact value of the local treatment with regard to the improvement found.

Its effect was more to be noticed, therefore, in the resistant type which had been treated so thoroughly with mercury, iodids, and salvarsan derivatives that one became almost hopeless of any further good resulting. Instillations have been continued over a period of months from the cessation of general treatment by salvarsan derivatives.

METHOD.—A few drops of a 1% solution of novarsenobillon derivatives in distilled water were dropped into the eyes usually at intervals of seven days, but in the later stage the interval has been fourteen days. The total number of cases treated was 20.

The immediate effect of instillation is a slight hyperemia at the corneal limbus. The patients usually complain only of slight irritation, but the lacrimal secretion is increased temporarily.

The patient is recumbent during the application, and is told to roll the eyeball about so as to get a uniform concentration of the drug, and the drops are added slowly, preferably until the dilatation of the circumcorneal vessels is well marked. The effects consist chiefly of some slight alleviation of the photophobia in the earlier cases, manifest after two or three weeks. Secondly, in the more resistant cases, in which intravenous injections have been abandoned, the corneal haze

gradually disappears and the substantia propria becomes comparatively clear. The cornea resumes its transparency first at the periphery, and usually the upper part of the cornea clears before the lower part. The disappearance of the corneal infiltration is, of course, associated with much improved vision, and one patient under this form of treatment can now thread with ease a very fine needle whereas previously she could scarcely detect hand movements. The effect in all cases has been to reduce the degree of corneal infiltration; and in relapsing cases to subdue the local inflammation.

ILL EFFECTS.—So far as can be ascertained no ill effects are produced. In one case only, a very difficult one, which had resisted all forms of treatment, a slight corneal ulcer formed and perforation occurred, but this might have happened quite apart from this form of treatment, and the patient made a good recovery, the cornea eventually clearing very well except for a small scar of the ulcer at the perforation site.

CONCLUSION.—This method is, I believe, a very valuable adjunct to the usual treatment; further, this method can be continued when further intravenous injections become dangerous.

It should, however, be used only as an adjunct to general systematic treatment of the disease.

It is interesting to speculate as to whether *Spirochaetae* are actually present in the cornea, and if so whether their persistence is due to the lack of blood supply to this tissue.

I wish to express my indebtedness to the courtesy of Dr. Clegg, who has specially assisted in this investigation and to the other members of the honorary staff who have also submitted their cases for this treatment.

TRATAMIENTOS DE LAS QUERATITIS INFECCIOSAS POR LAS VACUNAS

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LA BACTERIOTERAPIA EN OFTALMOLOGIA

Voy a tratar de un asunto completamente nueva en el terreno de la oftalmología, y de tal importancia, que creo, andando el tiempo, ha de constituir un verdadero tesoro para dicha especialidad.

El progreso que realiza la vacuna estafilocócica, y alcanza de día en día en el campo médico quirúrgico, no podía por menos de llamarme la atención y por ende ensayar un agente terapéutico que nos podía reportar ya como curativo, ya como coadyuvante, inmensos beneficios en la práctica de la oftalmología.

Todos sabemos los medios para combatir localmente los estragos causados por el estafilococo piógeno en el órgano de la visión; pero todos tampoco desconocemos la insuficiencia de estos medios en múltiples y determinados casos, como en panoftalmías, queratitis ulcerosas, dacriocistitis, blefaritis ulcerosas, etc., que ó bien se hace en unos casos necesaria la extirpación del órgano, y en otros, a pesar de los mejores medios empleados y la mayor constancia, se retarda la curación de un modo indefinido.

Sabemos que la experimentación clínica no ha demostrado todavía en las afecciones oculares la eficacia curativa de los sueros estafilocócico y estreptocócico (inmunización pasiva).

Por lo tanto un agente como la vacuna estafilocócica polivalente (inmunización activa), es decir, que obra excitando al organismo a la producción de anticuerpos en la totalidad del individuo, y al mismo tiempo que libran al organismo de lo que por la punción artificialmente le hemos introducido, llegan al proceso estafilocócico ocular y contribuyen a su curación, ha de ser, andando el tiempo y cuanto más se haya estudiado, jalón inestimable para la especialidad oftalmológica.

Los primeros vacunados fueron dos enfermos de panoftalmía, y como por su historia clínica se verá, en los dos se ha podido evitar la enucleación; mas en la mujer la reparación tomó tal incremento, que

fué aclarándose la córnea de tal manera, que ha llegado a distinguir visión de bultos; venía propuesta para enucleación de otra clínica, presentando fenómenos tan alarmantes, que yo también de no haber podido disponer de la vacuna, me hubiera visto precisado a recurrir al extremo antes dicho a fin de evitar la oftalmía simpática.

El tercer caso tratado con vacuna es un niño, a quien sólo incendié el saco lagrimal, raspando profundamente sus paredes, pero conservando el órgano, habiendo antes hecho una siembra en agar, y de la que se obtuvo una auto-vacuna. A la cuarta inyección había desaparecido el pus completamente, cicatrizando a los pocos días.

El examen bacteriológico llevado a cabo por el Dr. Mayoral dió un bacilo del grupo del diptérico, y que ha encontrado muchas veces en inflamaciones crónicas diversas, y sobre todo, en otitis y rinitis.

Los otros dos casos de raspados que corresponden al 16 y 18, en los que se emplean la auto-vacuna y vacuna estafilocócica, respectivamente, ambos cicatrizan rápidamente a los veintiuno y diez y nueve días de la aplicación de la vacuna.

En los demás casos ha sido la vacuna un poderoso coadyuvante, pues vemos se ha modificado muy satisfactoriamente la supuración, y en algunos casos por completo hasta el presente.

Por la rapidez con que se ha presentado la cicatrización en los tres casos de intervención, creo será un medio terapéutico no despreciable en las afecciones de las vías lagrimales, estando el beneficioso papel encomendado y digno de estudiarse a las auto-vacunas.

Cuarto caso. La enferma presenta dos úlceras, una que ocupa toda la parte supero-externa de la córnea, y otra la infero-externa de la misma, habiendo entre sus bordes una distancia mínima de dos milímetros por la parte central. La flogosis conjuntival es intensa, los dolores agudos y glerosis de la conjuntiva; a todo esto, como la enferma había perdido hace años el ojo congénere, por úlceras semejantes a las presentes, el estado de abatimiento es enorme. Hecha la siembra en agar, encuentra el Dr. Mayoral el estafilococo piógeno.

Después de la primera inyección, al día siguiente, hay un pequeño aumento de la flogosis y pus, por lo que la línea demarcativa de las ulceraciones avanza algo; avanzamiento muy compensado al otro día por la remisión, no sólo de los bordes ulcerados, sino de los trastornos subsiguientes a esta clase de queratitis, cesando también el estado de aplanamiento en que se hallaba la enferma, y se le práctica, con dos días de intervalo, una segunda inyección estafilocócica polivalente. Sólo se ha podido apreciar dos décimas de aumento en la temperatura,

y las queratitis marchan francamente en la curación. Sólo se emplean en días sucesivos dos inyecciones más hasta la curación definitiva.

Como huellas del proceso antes dicho, se aprecian en la córnea dos cicatrices sumamente tenues, y como se ha podido evitar la fusión de sus bordes por la parte central, la enferma queda con la visión perfecta.

Los casos 16° y 18° corresponden a una queratitis ulcerosa con hernia y a una queratitis perforante con hipopión, respectivamente; en ambos enfermos se ha inyectado la vacuna estafilocócica polivalente, y ambos han seguido idénticas fases que el caso primero expuesto, quedando la hernia reducida en el primero y cicatrizada la fístula corneal en el segundo.

Los tres casos de queratitis ulcerosa que he citado, como ven los lectores, son de lo más grave; y si bien se han tratado, como ya se supone, localmente, también he de advertir que estos medios fracasaron hasta que se aplicó la vacuna estafilocócica.

El último caso de úlcera serpiginosa de la córnea con hipopión, que ocupaba la tercera parte de la cámara anterior, rasando con la excavación ulcerosa, ha sido tratado por la vacuna pneumocócica del Laboratorio Municipal de Madrid.

Al día siguiente de la primera inyección acusa el enfermo haber sufrido durante la noche un fuerte dolor, como un latigazo. A la inspección macroscópica se observa una gran remisión del hipopión, el iris un poco sanguinolento y cierto rezumamiento de pus que, después de limpiado, deja ver una fístula en el centro de la úlcera, es decir que se trata de una paracentesis espontánea de la cámara anterior.

Al tercer día el hipopión, muy reducido, apenas se distingue en la parte inferior del ángulo corneal; al cuarto día, nueva inyección de vacuna pneumocócica; al quinto día ha desaparecido por completo el hipopión y la úlcera ha entrado en vías de franca cicatrización.

De no haberse verificado la paracentesis espontánea, claro está que toda la gloria recaería en la vacuna pneumocócica; pero como es bastante frecuente la terminación favorable de estos procesos por hechos análogos, ya de paracentesis natural ó provocada con el cuchillete lanceolar ó con un simple midriático, este hecho puedo restar algo de certidumbre, aunque la marcha ulterior, regresión rápida de todos los síntomas, la coloración del iris que recupera pronto su brillantez y la transparencia pupilar que se observa a los pocos días, hacen pensar que la vacuna no ha sido un factor despreciable en el presente caso.

Los cinco casos de blefaritis ciliar que a continuación expongo todos datan de muchos años de duración, y en algunos se remonta su comienzo a la edad infantil. En todos se ha comprobado los beneficiosos resultados de la vacuna estafilocócica desde las primeras inyecciones, y hasta el caso 8°, el más rebelde, y de síntomas más alarmantes, en cuya siembra se halló el *streptococo*, cedió a la auto-vacuna.

CASOS

1. Rafael López, veintisiete años, natural de Móstoles. Carretera de Getafe. Ingresó el 8 de Enero de 1915.

Diagnóstico: Úlcera infiltrada de toda la córnea ojo izquierdo, con síntomas de panoftalmía.

Terapéutica: Se le aplica la vacuna estafilocócica en los días 11, 14, 17 y 20, y se le da de alta el 31 del mismo mes.

Observaciones: A través del leucoma se percibe la coloración del iris; la temperatura no se ha elevado más que 3 décimas a la primera inyección.

2. Catalina Roncero Díaz, cuarenta y nueve años, natural de Toledo. Sombretería, 5. Ingresó el 14 de Enero de 1915.

Diagnóstico: Panoftalmía ojo izquierdo, propuesta ya para enucleación en otra clínica. La primera vacuna se efectúa el día de su entrada; el 17 se le aplica la segunda, y dos más en los días 20 y 23.

Observaciones: Desde la segunda vacuna cesan los dolores y se despeja la cornea por su parte superior; al terminar la cuarta, sólo presenta leucoma la mitad inferior corneal.

3. José María Vegas Perez, doce años, natural de Madrid. Pez, 1 principal.

Diagnóstico: Dacrio-cistitis ojo izquierdo; raspado profundo del saco lagrimal; a pesar de ello, hay alguna supuración, que desaparece a la tercera inyección de vacuna autógena, cicatrizando completamente después de la cuarta inyección.

4. Ana Maria Pizarro, sesenta años, natural de Puertollano. Ingresó el 17 de Febrero de 1915.

Diagnóstico: Queratitis ulcerosas ojo derecho. Se le aplica la vacuna estafilocócica en los días 21, 24 y 28 de Febrero y 4 y 8 de Marzo, dándosele el alta el 25 del mismo mes.

5. Priscila Pérez, diez años, natural de Villanueva del Campillo. Ingresa el 20 de Febrero de 1915.

Diagnóstico: Blefaro-conjuntivitis crónica de muchos años, a pesar de los tratamientos corrientes en esos casos, no se nota mejoría hasta la aplicación de la primera vacuna estafilocócica, el 12 de Marzo, aplicándola sucesivamente los días 16, 20, 25 y 31 del mismo mes.

Observaciones: En lugar de los bordes ciliares, rojos y desprovistos de

pestaña que presentaba la enferma a su ingreso en mi clínica, presentan en la actualidad su coloración normal, empezando a contarse algunas pestañas.

6. Benigno Galan, treinta y ocho años, natural de Madrid. Miraelso, no. 4. Entró el 8 de Marzo de 1915.

Diagnóstico: Blefaro-conjuntivitis antigua, desde que era niño. Se le aplica la vacuna estafilocócica en los días 12, 16, 20, 26 y 31 de marzo y 8 de Abril, habiendo mejorado notablemente.

7. Hermenegilda dle Barrio, treinta y siete años, natural de Segovia. San Vicente, 38. Entró el 20 de Diciembre de 1914.

Diagnóstico: Dacrio-cistitis ojo izquierdo; se la trata con sondas é irrigaciones desinfectantes, a pesar de lo cual no cesa del todo la supuración. Se le inyecta la primera vacuna estafilocócica el 12 de Marzo de 1915, y se le vuelve aplicar en los días 16, 24 y 31 del mismo. En la actualidad ha cesado la supuración; sólo hay excreción del líquido mucoso.

8. Mercedes Iglesias Taboada, treinta y un años, natural de Toledo Divino Pastor, 1.

Diagnóstico: Blefaro-conjuntivitis crónica de muchos años, pérdida absoluta de las pestañas y escoriaciones que, naciendo del borde ciliar se extienden por la epidermis palpebral. Se le aplica la vacuna estafilocócica en los días 13, 17, 20, 24 y 31 de Marzo, habiéndose modificado bastante, pero no lo debido, por lo cual se le hace una vacuna autógena en la que se halla el estreptococo, y con cuya aplicación se logra el resultado apetecido.

9. Francisca Recio Guerra, veinticuatro años, natural de Santa Cruz del Retamar. Corredera Baja, 5.

Diagnóstico: Blefaro-conjuntivitis ulcerosa, habiendo sido ya operada con éxito de triquiasis; los bordes escoriados y sin pestañas; se le aplica la vacuna estafilocócica en los días 13, 16, 20 y 31 de Marzo, y 3 y 6 de Abril; en el presente se observa una coloración normal en los bordes ciliares, y acusa la enferma una comodidad nunca sentida.

10. Amalio Quiroga, 18 años, natural de Villanueva de la Torre. Luisa Fernanda, 14.

Diagnóstico: Blefaro-conjuntivitis eczematosa, presentando un eczema muy extenso en las cejas y la frente, que ha resistido a cuantos tratamientos se han ensayado en él. Se le aplica la vacuna estafilocócica en los días 13, 16, 20, 24, y 31 de Marzo, habiendo desaparecido, no sólo el eczema ciliar, sino también el frontal.

11. José Zamora Mendez, natural de Madrid, Buenavista, 35.

Diagnóstico: Dacri-cistitis doble: a pesar de haberse empleado las sondas de mayor calibre é irrigaciones desinfectantes, no cesa la supuración. Se le aplica la vacuna estafilocócica en los días 13, 16, 23 y 31 de Marzo, y 3 de Abril, habiendo desaparecido completamente el pus del ojo derecho y siendo necesario el empleo de una auto-vacuna para conseguir la extinción del pus del ojo izquierdo.

12. Benita Gonzalez, treinta y siete años, natural de Fuencarral. Ingreso el 24 de Febrero de 1915.

Diagnóstico: Dacrio-cistitis doble; tratamiento: sondas é irrigaciones antisépticas; a pesar de ello, el pus no desaparece. Se le aplica la vacuna estafilocócica en los días 14, 18, 22 y 26 de Marzo, habiendo desaparecido el pus hasta el presente.

13. Ramón Herranz, cincuenta y cinco años, natural de Moralejo. (Segovia). San José, 2.

Diagnóstico: Dacrio-cistitis ojo derecho; a pesar de sondársele mucho tiempo, no desaparece el pus. Se le aplica la vacuna estafilocócica polivalente en los días 14, 20, 25 y 30 de Marzo; en le presente ha cesado la supuración.

14. Hipólita Vidal, treinta y dos años, natural de Recas (Toledo). Plaza de Salmeron, 4.

Diagnóstico: Dacrio-cistitis doble; la enferma se niega tanto a las sondas como a la operación. Se le aplica la vacuna estafilocócica en los días 15, 18, 24 y 30 de Marzo y 8 de Abril, habiendo cesado la supuración pero conservando cierta rubicundez y mucosidad que, dada la naturaleza de la enfermedad y el poco tiempo de tratamiento, no permiten emitir un juicio satisfactorio y definitivo.

15. Rosalia Pastrana Torre, veintidós años, natural de Madriedejos.

Diagnóstico: Ulcera perforante de la córnea, con enturbiamiento de toda ella, glerosis conjuntival y fuertes dolores; en vista del incremento que iba tomando la supuración, a pesar de los múltiples lavados antisépticos, se le aplica la vacuna estafilocócica polivalente en los días 22, 25, y 30 de Marzo, y 3 y 9 de Abril. La enferma es dada de alta el 14 de Abril, con una cicatriz apenas perceptible y una buena visión.

16. Agapita Palafox, treinta y dos años. Guadalajara.

Diagnóstico: Dacrio-cistitis, ojo izquierdo, en el que se le práctica un profundo raspado; y como continua la supuración, se hace una siembra en la que se halla el estafilococo, del que se obtiene una autovacuna que se le aplica el 27 y 31 de Marzo y 4 y 11 de Abril, habiéndose extinguido por completo la supuración, con conservación del órgano y de su función.

17. Nominando Lopez Villar, cincuenta y tres años, natural de Juncos (Toledo).

Diagnóstico: Ulcera herniada con hipopión. Se le aplica la vacuna estafilocócica el 31 de Marzo y 3 y 6 de Abril; se ha podido reducir la hernia, y habiendo cicatrizado completamente ha recuperado una buena visión.

18. Ginés Monllor Domenech, treinta y nueve años, natural de Madrid. Plaza del Rastro, 9.

Diagnóstico: Dacrio-cistitis ojo izquierdo; raspado del saco lagrimal. Como hay supuración, se le aplica la vacuna estafilocócica en los días 5, 10,

13, y 16 de Abril. A los pocos días de la primera inyección desapareció el pus y la cicatrización se presenta franca.

19. Cirilo de la Torre, cincuenta años, natural de Valdearenas. (Gualajara.)

Diagnóstico: Ulcera serpiginosa, con grande hipopión, en el ojo derecho; se le aplica la vacuna pneumocócica en los días 9, 12, y 15 de Abril. Al día siguiente, a la primera inyección, sobreviene una paracentesis espontánea; iris sanguinolento y cesación de dolores; en las inyecciones siguientes desaparece por completo el hipopión; la pupila se presenta completamente negra y redonde; hay una excelente visión.

MARCHA

En las enfermedades crónicas, como dacrio-cistitis, eczemas palpebral blefaritis ciliar, etc., puede empezarse con dosis de 0.50, 1.00 y 1.50 de vacuna estafilocócica polivalente, siendo en las autovacunas la mitad de las dosis antedichas, siempre teniendo en cuenta el peso del individuo y en las afecciones agudas, oftalmías y queratitis ulcerosas, a pesar de que hay que tener en cuenta la fase negativa, nosotros hemos empleado las mismas dosis elevadas, sin que la pequeña agravación de trastornos locales que se nota en esta haya sido de gran duración; en los presentes casos nunca ha llegado a las treinta horas; y en cuánto a los fenómenos generales, sólo hemos podido apreciar unas décimas de elevación de la temperatura.

Si esta fase negativa resulta algo intensa, hay que retardar algo más la segunda punción y no aumentar más la dosis anterior.

RESUMEN

Cinco blefaritis ciliares de las más rebeldes y antiguas, curadas. Se ha empleado la vacuna estafilocócica y streptocócica autógena.

Tres casos de raspado de saco lagrimal con conservación de órgano y función.

En los demás tratados por el sondaje se ha extinguido el pus hasta el presente.

Se ha empleado la vacuna estafilocócica y auto-vacunas de gérmenes diversos.

Dos panoftalmías, en las que se ha podido evitar la enucleación y conservación, y alguna agudeza visual en una.

Pero donde las vacunas, como se ve por los casos anteriormente citados, han alcanzado un importante y brillantísimo papel, ha sido en las afecciones corneales, precisamente las más temidas.

La rapidez de la cicatrización que hemos podido observar, la falta de huellas cicatrizales y la conservación funcional, ponen a las vacunas, en primer lugar, en el tratamiento de las queratitis ulcerosas.

Se ha empleado la vacuna estafilocócica y pneumocócica.

Por lo tanto se han ensayado en esta experiencia las vacunas estafilocócica polivalente, la pneumocócica, y las auto-vacunas streptocócica, estafilocócica y una del grupo diftérico.

En oftalmología, como en las demás ramas de la Medicina, se impone hoy el diagnóstico bacteriológico de los procesos infectivos y, sobre todo, los localizados ó crónicos, pues la bacterioterapia ha ensanchado enormemente el campo de los tratamientos específicos; el tratamiento bacterioterápico es compatible con los demás, locales ó generales, que la experiencia ha demostrado ser los mejores, y los ayuda haciendo colaborar a la totalidad del organismo en el proceso de curación local.

Las vacunas polivalentes se emplearan con preferencia, por ser de más cómoda preparación y, por lo tanto, más económicas; pero cuando el proceso es poli-microbiano; cuando está causado, por gérmenes contra los que no hay preparada vacuna polivalente ó de depósito, y cuando ha fracasado la vacuna polivalente, deberá recurrirse a las auto-vacunas.

TRECE CASOS DE ULCERA SUPURATIVA DE LA CORNEA TRATADOS CON VACUNA ESTAFILOCÓCICA

Las queratitis no supurativas, tanto superficiales como profundas, ceden por lo general la mayoría de las veces con un tratamiento apropiado, pero no pasan las cosas en la misma forma cuando se trata de queratitis supurativas, que dividiremos en tres grupos: úlceras, accesos, y queratitis por desnutrición.

Sólo voy a tratar aquí de las primeras.

De entre estas descontaremos las queratitis secundarias debidas al gonococo, difteria y tracoma, que responden a una terapéutica ya estudiada.

Nos restan cuatro especies de queratitis supurativas que son en el orden de frecuencia las siguientes: (1) Úlceras debidas al estafilococo piógeno, más raramente al estreptococo; (2) úlceras serpiginosas, típica o atípica, pneumocócica; (3) queratomicosis aspergilar; (4) úlceras liquidante, diplobacilo, de Petit de Rouen, de la que sólo se han reunido contadas observaciones.

Tanto la queratitis liquidante como la queratomicosis aspergilar,

debida al "aspergillus fumigatus," su paso por las clínicas es por extremo raro, tanto que pueden pasar años sin que el especialista observe un solo caso.

La queratomicosis aspergilar cede por lo general a un concienzudo raspado coadyuvado por los medios desinfectantes ordinarios y la queratitis liquidante es de esperar que ceda a la autovacuna.

Nos quedan, pues, como las más frecuentes, las queratitis estafilocócica, estreptocócica, y pneumocócica; pero estas dos últimas en una proporción por demás inferior.

De este análisis se deduce que la úlcera estafilocócica es la más frecuente de todas; es la que tenemos cotidianamente entre manos, y como su gravedad es extrema, de aquí la importancia de un tratamiento como el que tengo el honor de exponer, con el cual he alcanzado siempre hasta el presente resultados satisfactorios.

La queratitis estafilocócica queda bien deslindada por sus caracteres clínicos de la úlcera pneumocócica, a lo menos en sus comienzos; pero no así de la queratitis estreptocócica, si bien ésta es sumamente rara.

Por esto se impone el examen bacteriológico, pues puede darse también el caso de asociarse ambas bacterias en el mismo proceso. Esto, no obstante, siempre que sospechemos por los caracteres clínicos un caso de úlcera estafilocócica, a más de obtener varias siembras en caldo aerobio y anaerobio en agar y suero debe practicarse desde luego la inyección de vacuna estafilocócica por ser ésta inofensiva, y que casi siempre el examen bacteriológico comprobará el aserto, en cuyo caso habremos ganado un tiempo inapreciable, y en el caso de hallarse otros gérmenes, puede continuarse el tratamiento con las autovacunas elaboradas con dichas bacterias.

Los trece casos que a continuación expongo de queratitis supurativas en todos la ulceración alcanzó grandes dimensiones, presentando abundante hipopión, habiendo por lo tanto llegado a mi clínica en un estado de suma gravedad, y en los cuales se habían empleado los medios ordinarios sin resultados. En dichos casos hay que anotar las siguientes observaciones: El caso séptimo, recomendado por el Dr. Rey Becerra, corresponde a un enfermo que además de presentar una extensa ulceración corneal, con hipopión, está afecto de dacriocistitis con abundante supuración, y al que practiqué en el acto la extirpación del saco lagrimal; no obstante la ulceración y el hipopión siguen progresando, a pesar de una rigurosa antisepsia, siendo necesario aplicar la vacuna al segundo día de operado; la mejoría se

observa rápidamente, y el enfermo ve hoy regularmente gracias a una iridectomía.

El caso undécimo, muy parecido al anterior, pues presenta también la enferma úlcera con hipopión y dacriocistitis; pero en este caso se ha podido conservar la visión sin necesidad de recurrir a la iridectomía, pues se le aplicó la vacuna desde el primer momento antes de operar el saco, y tanto el proceso corneal como lagrimal cicatrizaron en breve tiempo.

El caso noveno, el más grave de todos, lo recomienda el Dr. Bajo; la úlcera se extiende casi hasta los límites corneales y el hipopión tapa completamente el iris; después de la aplicación de la vacuna cede tanto la ulceración como el hipopión hasta la desaparición de ésta; queda un leucoma adherente, pero deja sitio para una iridectomía bastante extensa.

El caso duodécimo recomendado por el doctor Santacana, que a pesar de presentar una ulceración central y extensísima con hipopión enorme, apenas ha dejado huella cicatrizal, tanto que la enferma ve regularmente no obstante el leucoma central, por ser este muy diáfano.

CASOS

1. Socorro Patón, de cuarenta y dos años, natural de Valdepeñas, ingresa el 31 de Mayo del corriente año, afecta de úlcera supurativa con hipopión. Se le aplicaron tres inyecciones de vacuna estafilocócica polivalente del Laboratorio Municipal; leucoma reducido en la parte inferior de la córnea, bastante transparente.

2. Rufina García, de treinta años, de Casatejada (Cáceres), ingresó el 2 de Junio con úlcera supurativa con hipopión del ojo izquierdo. Se le aplicó tres veces la vacuna estafilocócica polivalente. Leucoma muy diáfano.

3. Pablo Berlanga, de cincuenta y siete años, de Vallecas, ingresa el 2 de Junio, presentando en su ojo derecho una extensísima úlcera con abundante hipopión; se le aplicaron cinco inyecciones de vacuna estafilocócica polivalente. Leucoma adherente. Iridectomía óptica. Regular visión.

4. Pedro Sanz, de sesenta y seis años, de Cedillo de la Torre (Segovia), ingresa el 16 de Julio con úlcera traumática con hipopión.

5. Juana Viñuelo, de cincuenta y seis años, de Puebla de Veleña (Guadalajara), ingresa el 29 de Julio presentando una úlcera con hipopión ojo izquierdo; se le aplicó la vacuna estafilocócica en los días 24, 26 y 29 de Julio y 2 y 5 de Agosto. Leucoma algo extenso pero muy transparente.

6. Ramón Peral de cincuenta años de edad, de Navas del Rey (Madrid), ingresa el 27 de Julio afecto de úlcera supurativa con hipopión en su ojo izquierdo; se le aplica la vacuna estafilocócica en los días 28 de Julio, 1, 3, y 6 de Agosto. El leucoma ocupa el cuarto externo de la córnea.

7. Gervaso Sanchez de cuarentay ocho años de Espinosa del Rey (Toledo), ingresa el 30 de Julio afecto de dacriocistitis y úlcera con hipopión en su ojo izquierdo; se le aplica la vacuna estafilocócica en los días 31 de Julio, y 2, 4, 7, 11, y 16 de Agosto. Leucoma adherente: Iridectomía.

8. Esteban Uceda, de cincuenta y seis años, de Brunete (Madrid), ingresa el 11 de Agosto con una úlcera central con hipopión en su ojo izquierdo; se le aplicó la vacuna estafilocócica en los días 12, 16, 18 y 21 del mismo mes: Leucoma muy transparente.

9. Narciso Luengo, de cuarenta y nueve años, de Calzada de Orepesa (Toledo), ingresa el 18 de Agosto presentando en su ojo izquierdo una úlcera extensísima con abundante hipopión; se le aplican cinco inyecciones de vacuna estafilocócica: Leucoma tercio inferior corneal.

10. Rosario Arellano de veintocho años, de Fuensalida (Toledo), ingresa el 31 de Agosto presentando una úlcera con hipopión en su ojo izquierdo; se le aplicó tres veces la vacuna estafilocócica: Leucoma transparente.

11. Alejandra Dueñas, de cincuenta años, de Yébenes (Toledo), ingresa el 4 de Septiembre afecta de dacriocistitis y úlcera supurativa con hipopión de su ojo izquierdo; se le aplicó la vacuna en los días 6, 8, 10, 13, y 15 del mismo: Leucoma inferior y transparente.

12. Adela de la Peña, cuarenta y ocho años, de Zarzalejo (Madrid), ingresó el 10 de Septiembre afecta en su ojo derecho de úlcera central con hipopión; se le aplicó tres veces la vacuna estafilocócica polivalente: Leucoma muy diáfano.

13. Juliana Serrano, veintiocho años, de Palencia, ingresó el 11 de Septiembre presentando en su ojo izquierdo una úlcera supurativa con hipopión; se le aplica tres veces la vacuna estafilocócica polivalente: Leucoma muy transparente.

RESUMEN

Del estudio de los precedentes casos he podido deducir las conclusiones siguientes:

1. La bondad del procedimiento; pues en los trece casos de úlcera corneal supurativa a cual más graves que he presentado, en todos se ha podido conservar la visión; y si a estos trece casos unimos los cuatro que expuse en mi anterior artículo sobre la bacterioterapia en oftalmología, suman diez y siete observaciones de queratitis supurativa en las que ha triunfado el tratamiento por la vacuna, cuando ya habían fracasado los demás medios hasta aquí conocidos.

2. Que a pesar de las dimensiones exorbitantes de la ulceración que han presentado la mayoría de los citados casos, la extensión del leucoma no es proporcional a dichas dimensiones, pues es más pequeño y más transparente que los leucomas que quedan en las úlceras tratadas por los medios ordinarios.

3. Que si bien en la mayoría de los casos se nota la mejoría desde el día siguiente de la primera inyección, hay algunos en que esta mejoría es más tardía, sobre todo cuando se trata de procesos de extrema gravedad, habiendo podido observar en dichos casos la regresión de los síntomas cuando más inminente parecía el peligro; regresión que se fué acentuando hasta quedar el ojo con una regular visión, por lo que recomiendo perseverar, a pesar de los fenómenos alarmantes que presente el proceso hasta la quinta o la sexta inyección.

Una rigurosa antisepsia local completará el tratamiento.

Por lo tanto estamos en presencia de un agente terapéutico, el más eficaz en el tratamiento de las queratitis supurativas. El mejor, por la diafanidad de las huellas cicatrizales que se alcanza con su empleo, y que es además completamente inofensivo.

APLICACIONES PRÁCTICAS DE LA INMUNIDAD EN OFTALMOLOGÍA

No es nuestro objeto hacer un estudio completo de la inmunidad en Oftalmología, pero si creemos conveniente exponer los principios que han guiado nuestros estudios de bacterioterapia oftálmica y las reglas que seguimos en la aplicación de las vacunas, nuevo recurso terapéutico que necesita manejar bien el oculista.

La lectura del admirable trabajo del Dr. Juan Campos Fillol, Profesor auxiliar de la Facultad de Medicina de Valencia, titulado "Investigaciones acerca de la cantidad de anticuerpos en el suero de la sangre y en los humores del ojo" demuestra cuanta atención deben dedicar al estudio de la sueroterapia y bacterioterapia especial oftalmológica los que practican esta especialidad médica, y aun los médicos generales.

El Dr. Juan Campos ha demostrado con sus importantes trabajos: (1) Que los anticuerpos se encuentran en el humor acuoso y vítreo en cantidad muy inferior a la del suero sanguíneo, proximamente en la relación de 1 por 100; (2) que las inyecciones subconjuntivales y otros estímulos externos del globo ocular acrecientan la cantidad de anticuerpos contenidos en los humores del ojo; (3) la paracentesis corneal eleva la proporción de anticuerpos del humor acuoso entre cuatro y cincuenta veces. En consecuencia con estos datos, aconseja la aplicación directa de los sueros antitóxicos y bacteriolísicos, cuando necesiten emplearse para combatir infecciones oculares, y espera que "la sueroterapia específica y paraespecífica, la autosueroterapia y la aplicación de vacunas, lo mismo profilácticas que curativas, enri-

quezcan el arsenal terapéutico de la Oftalmología, proporcionando brillantes éxitos."

Suscribimos en absoluto la idea del Dr. Campos, referente a la aplicación directa de los sueros terapéuticos al ojo, por ser el mejor medio de llevar al foco del mal en máxima concentración las substancias defensivas que los sueros contienen. La instalación frecuente de suero en la conjuntiva cuando se trate de procesos superficiales de la córnea; la inyección subconjuntival y hasta la introducción directa en la cámara anterior del ojo, son los medios que nos permiten obtener el fin deseado.

Cuando en vez de suero se emplean vacunas; cuando no se inyectan substancias defensivas, sino, por el contrario, tóxicas, que obligan a reaccionar al organismo, haciendo que él prepare los anticuerpos que necesita para destruir los gérmenes que le atacan y neutralizar sus toxinas, entonces hay que inyectarlas lo más lejos posible del foco, en el tejido celular subcutáneo ó en las venas.

La inyección de vacunas obliga a la totalidad del organismo a colaborar en la defensa que realizan los elementos atacados del ojo y los que aporta la sangre, y se fuerza la producción de substancias defensivas que permiten yugular el proceso local.

La inyección metódica de vacunas provoca la aparición de anticuerpos en el suero de la sangre, principalmente, por reacción de los elementos del tejido conjuntivo: sangre, ganglios linfáticos, bazo y médula ósea, excitados por las substancias microbianas que las vacunas contienen, y que no se realiza, o, de verificarse, es mucho menos intensa cuando el individuo que tiene una infección ocular no se somete a este tratamiento.

Según el Dr. Campos, la cantidad de anticuerpos elaborados por la totalidad del organismo que pasan del plasma hemático a los humores del ojo, es muy inferior a la que este contiene, é indica que artificios podemos utilizar para aumentar dicho paso; pero hay que tener en cuenta que nuestro compañero ha experimentado en ojos normales. Ha visto que los estímulos que sobre el globo ocular se realizan elevan la proporción de anticuerpos de los humores oculares; teniendo en cuenta estos datos y las observaciones clínicas que hemos publicado sobre bacterioterapia de las queratitis supuradas, creemos que el estímulo que realiza la infección ocular es suficiente para atraer al ojo los anticuerpos hemáticos en cantidad suficiente para neutralizar la acción de los gérmenes.

La paracentesis de la cámara anterior es, según el doctor Campos,

el más poderoso recurso de que disponemos para enriquecer en anticuerpos hemáticos el humor acuoso. Nosotros hemos podido evitar la paracentesis con la bacterioterapia en casos en que parecía indicada; no obstante, la reputamos como un excelente recurso cuando se acumula gran cantidad de pus en la cámara anterior, pues con ello se libra al ojo de las sustancias tóxicas microbianas, y de la tripsina leucocitaria que queda libre al destruirse los polinucleares, y cuya acción sobre los tejidos oculares es nefasta. Además, la paracentesis obra aportando anticuerpos y elevando el poder antitripsico del humor acuoso.

Quizá se presente algún caso en el que la relativa dificultad que señala el Dr. Campos para el paso de los anticuerpos de la sangre a los medios del ojo, dificulte obtener el máximum de beneficios que puede reportar el empleo de las vacunas; es decir, que a pesar de la intensa producción de sustancias defensivas por el organismo, provocada por la inyección de vacuna, pasen en escasa cantidad al foco ocular, en el que han de cumplir su acción anti-infecciosa. En estos casos, estaría plenamente indicada la autosueroterapia de que habla el Dr. Campos; diez o doce horas después de extraer al enfermo por punción venosa algunos c.c. de sangre, se podría inyectar su suero, y con el los anticuerpos debajo de la conjuntiva ó en la cámara anterior del ojo, sin miedo a ocasionar reacciones séricas, ya que procede del propio individuo.

De lo expuesto se deduce que la inmunización activa terapéutica que realizan las vacunas es perfectamente compatible con los medios de acción local, cuya eficacia ha comprobado la experiencia y las molestias que ocasiona al enfermo, infinitamente menores que las ventajas que sobre la enfermedad se obtienen.

El empleo de las vacunas en Oftalmología se rige por las reglas generales de bacterioterapia; ante todo, es necesario establecer el diagnóstico etiológico de la infección ocular. Unas veces, este diagnóstico se hará por los medios de que dispone la bacteriología; pero en otras, los caracteres clínicos permitirán suponer de que gérmenes se trata.

Por lo que se refiere a las infecciones supuradas de la córnea, que son los procesos oculares en que hemos estudiado prácticamente la bacterioterapia, diremos que la gonocócica comienza por una infiltración grisácea y limitada de la córnea, que abandonada a si misma, se extiende rápidamente a la totalidad de la membrana; como caranteres

diferenciales, señalaremos lo enorme del glérosis conjuntival y lo abundante de la supuración típica de este proceso.

En la queratitis tracomatosa la ulceración es casi siempre múltiple y más o menos profunda, según la mayor o menor destrucción de la córnea por la lesión tracomatosa, siendo los síntomas diferenciales el pannus, las granulaciones ó las cicatrices granulosas.

En las queratitis diftérica, la ulceración se extiende rápidamente y va acompañada de iritis é hipopión.

Estos caracteres clínicos, unidos a los antecedentes, nos permitirán casi siempre suponer la etiología de esta clase de queratitis.

De las restantes queratitis supurativas, la más frecuente es la causada por el estafilococo piógeno, siguiendo en orden de frecuencia la queratitis neumocócica, estreptocócica, diplobacilar, aspergilar y licuante.

Por orden de frecuencia, las queratitis debidas al estafilococo y al pneumococo son las más importantes, siendo muy distinto el síndrome de unas y otras.

La úlcera serpigiosa, semilunar, de base infiltrada, ó sea la pneumocócica, se presenta con el característico borde en forma sigmoidea; este está elevado y socavado en fondo de saco, limpiándose y alisándose los bordes situados en el lado opuesto que se recubre de epitelio sin formación de vasos.

La queratitis estafilocócica se asemeja, por su aspecto, a las ulceraciones simples no sépticas, de las que la diferencian lo rápido del crecimiento y las tumultuosas manifestaciones que la acompañan. La infiltración ocupa toda la pérdida de substancia, al contrario de la pneumocócica (*ulcus serpens*), que avanza sólo por su borde sigmoideo.

El hipopión, inyección ciliar, dolor, etc., síntomas que acompañan a esta clase de queratitis, no lo señalo, para no distraer los caracteres verdaderamente diferenciales.

En la queratitis debida al estreptococo, los síntomas son muy parecidos a la producida por el estafilococo, de la que sólo difiere por su marcada propensión a la perforación, pues la infiltración invade desde los primeros momentos las capas corneales profundas.

En la queratitis debida al diplobacilo de Morax, la infiltración es marginal y grisácea, cede pronto al sulfato de zinc al 3 por 100. La bacilar de Weekes puede ser central, es superficial, y grisácea, hay secreción y cede a los preparados argénticos.

La queratomicosis, debida al *aspergillus fumigatus*, presenta una

infiltración corneal reducida al principio, análoga a una flictena, rodeado de bordes limpios y cortantes. La queratitis licuante, debida al diplobacilo de Petit (Rouen), es muy parecida a la úlcera serpigginosa superficial o sea la típica, pero se diferencia de ésta por la característica de ser completamente indolora.

Conocido el germen causante de la lesión que necesitamos combatir, podemos emplear una autovacuna, o sea una vacuna hecha con los propios gérmenes aislados en el enfermo, o vacunas ya preparadas, polivalentes ó de depósito. Las autovacunas son más eficaces que las de depósito ó comerciales, pero su preparación no es posible en todos los casos, y siempre es más costosa. Las vacunas comerciales son suficientes en la inmensa mayoría de los casos, como hemos tenido ocasión de demostrar en nuestros trabajos, pues casi todas las observaciones son casos tratados con vacuna polivalente.

Las inyecciones de vacuna provocan reacciones que es necesario observar atentamente, pues ellas son los datos que nos sirven para dirigir el tratamiento; estas reacciones son: local, general y focal.

Reacción local es la que se observa en el sitio en que la vacuna se inyecta, siempre de naturaleza inflamatoria, más ó menos intensa, según la dosis de vacuna inyectada y las condiciones individuales.

La reacción general consiste en malestar, inapetencia, insomnio, frecuencia de pulso, y sobre todo, elevación de temperatura, que habrá que observar con ayuda del termómetro.

La reacción focal nos interesa muy especialmente, pues dada la delicadeza del órgano en que la lesión asienta, una reacción excesiva en ella puede ser perjudicial. Nosotros hemos tenido la fortuna de no observar reacciones focales perjudiciales, pues hemos procedido con cautela en la elevación progresiva de la dosis, sobre todo en la primera inyección que sirve para tantear la susceptibilidad del sujeto; en la primera inyección hemos utilizado siempre dosis pequeñas, inferiores a las que aconseja el preparador de la vacuna en las instrucciones que la acompañan.

Cuando la inyección produce reacción local o general muy fuerte, y con mayor motivo si se observa reacción local, la dosis empleada es grande, y en la inyección siguiente no se aumentará la dosis; se repetirá la que resulte fuerte hasta que el enfermo la tolere bien.

Procediendo como queda dicho, hemos obtenido los brillantes resultados publicados en los trabajos precedentes, y que demuestran que no es hoy especialista completo quien no sepa servirse de la bacterioterapia, y muy especialmente para combatir la queratitis

supurada, afecciones oculares que con mayor frecuencia conducen a la pérdida de la visión.

En fecha 1 de Junio de 1915, registramos 31 casos de queratitis supurada, todos ellos graves, en los que habían fracasado los medios terapéuticos habituales, y que la bacterioterapia nos permitió curar rápidamente; a pesar de las enormes dimensiones de la ulceración que presentaban la mayoría de los citados casos, la extensión del leucoma, que se produjo al curar no es proporcional a dichas dimensiones: es más pequeño y transparente que los producidos después de úlceras tratadas con los medios ordinarios.

Hoy, en el día de la fecha, la estadística se remonta a 472 casos tratados por las vacunas y en los que se ha comprobado los resultados satisfactorios precitados.

CARCINOMA OF THE CHOROID

DR. ALLEN GREENWOOD

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Metastatic carcinoma of the choroid is such an infrequent manifestation of a general carcinosis that the chance to see and follow more than one case but rarely falls to the lot of an ophthalmologist. The opportunity which has come to the author to follow four cases makes it seem worth while to publish them.

The first case, that of a man, W. G. C., was published fully in the Boston Medical and Surgical Journal by Greenwood and Southard¹ in 1903, including the results of a postmortem examination and an exhaustive microscopic examination of all metastatic growths. At that time 29 cases of metastatic carcinoma of the choroid had been reported. Later, Oatman,² Parsons,³ and Krukenberg⁴ brought the number of cases up to 41 and in 1908 Suker and Grosvenor⁵ brought the number up to 64. Since then a number have been reported.

On reference to the author's report of his first case, it will be seen that the patient came to the hospital on account of acute glaucoma

¹ Greenwood and Southard: Boston Med. and Surg. Jour., 1903.

² Oatman: Am. Jour. Med. Sci., March, 1903.

³ Parsons: Royal London Ophth. Hosp. Report, xv, 3.

⁴ Krukenberg: Manz-Sattler Festschr., Klin. Monatsbl. f. Augenh., 1903, xli 145.

⁵ Suker and Grosvenor: Trans. Am. Acad. Ophth. and Oto-Laryn., August, 1908.

in the left eye. The fundus, after the glaucoma had been relieved by miotics, showed a separation of the retina in the upper temporal quadrant, covering a flat, grayish-white mass. A diagnosis was made of sarcoma of the choroid, but the eye was not removed as it was suspected that other organs were involved, particularly the liver. The ocular pain was easily controlled by cocain and miotics. The general examination of this patient had shown no evidence of any tumors in other parts of the body, except the probability of an involvement of the liver, which was enlarged. Prior to his death the patient became mentally incompetent, suggesting an involvement of the brain. The autopsy findings would indicate the prostate as the original source of the general carcinosis. The organs particularly involved in the general disease were the lungs, pleura, peribronchial lymph-glands, the liver, the brain, and the eye. In this case, as in one other, the eye condition was the first to call attention to the possibility of disease in other parts of the body.

CASE II.—Miss J. D., aged forty-eight, cook. Came under observation April 12, 1908, with the history that the sight in the left eye had been dim for several months and lately had grown very much worse. Right eye normal. Vision = 20/20 and fundus normal. Left eye had a vision of hand motions only in outer field. Fundus showed a retinal separation in the outer part extending up to the disc, and including most of the upper and lower part as well. Through the separated retina 3 d. d. temporally from the disc could be seen a white mass elevated centrally about 3 d. d., which gradually flattened out in all directions. The tumor was quite white in the most elevated portion, giving the appearance as though a disc of some substance like cheese had been inserted under the retina. Immediately the similarity of the fundus picture to that seen in Case I came to mind and questioning the patient brought out the history that for a year there had been a hard lump in the left breast and she had become very short of breath. She had seen no physician so was sent to one who reported cancer of left breast, involving left lung and the axillary glands; therefore, unoperable. Seen again August 4, 1908, complaining that the right eye had a spot of dimness. Right eye examination showed up and out from the disc a whitish, flat mass about 2 d. d. in size with retina over it elevated 1 D. This tumor beneath the retina corresponded in all but size to the one seen in the left eye in April. The left eye showed at this time complete retinal separation and opaque striæ in lens. Tension was normal in both eyes. I next saw the patient in consultation at the Waltham Hospital in October, 1908, she having been taken there on account of her weakened general and mental condition. I found her semi-conscious and incoherent. The tumor mass in the right eye had increased

to more than double its former size and was elevated at its highest point 4 d. d. and extended to the disc.

The left eye showed a retinal separation up against a cloudy lens. The patient died several weeks later from cerebral involvement.

CASE III.—Mrs. J. S., aged sixty. Seen September 12, 1912, at her home as she was too feeble to come to the office. Patient gave a history of vision failing for several months beginning first in the left eye and shortly after in the right. Two years before had been through an amputation of the right breast and dissection of the axilla for carcinoma. Had been losing strength and had a harsh, dry cough with much shortness of breath. Examination showed right eye fundus with a flat retinal separation out and down. Retina elevated 3 d. d. about 4 d. d. down and out from disc with the elevation growing less as one looked toward the disc, or peripherally. A white discoid tumor mass could be seen through the overlying retina, but no independent vessels were to be seen and no inflammatory reaction was present. Left eye fundus showed extensive separation of the retina from the disc outward and extending somewhat to the nasal side above and below the disc. The lower part of the separation was billowed forward by exudate. Up and out there could be seen, through the overlying retina, a grayish-white mass, somewhat uneven, the highest elevation being about 4 D. This mass extended from the temporal side of the disc, where it was very thin, outward beyond the equator. There was no independent vessel formation on the mass. The pupils were fairly large but tension not above normal. The daily use of pilocarpin was advised with the hope of preventing hypertension and pain. The patient died three months later with evidences of cerebral involvement and practically blind, but without the eyes becoming painful.

CASE IV.—Mrs. A. E. H., aged thirty-six. Seen November 7, 1921, complaining that "something seems to come frequently before the right eye so that print blurs and there is a constant tendency to rub something from the right eye." Vision O. U. = 20/30. Patient is slightly myopic. Examination of fundus, right eye, revealed a very flat discoid subretinal mass located down and out from the macula. It was round and 3 d. d. in size and the upper inner edge reached to lower border of the macula. It was quite white, with the center whiter than toward the periphery. No independent vessel formation was seen and no measurable elevation of the retina. No evidence of inflammatory reaction to its presence. The visual field showed a partial scotoma up and in. A more extensive examination of the field was not attempted at this time on account of the patient's poor physical condition. From these appearances, which were similar to those in Cases I, II, and III, a diagnosis of carcinoma of the choroid was made and then the history was obtained of operations for mammary carcinoma April 2, 1920, and October 4, 1920, at the Massachusetts General Hospital. A few days later an x-ray examination at the hospital showed extensive involvement of the left lung. Efforts were made to have additional examinations but this has not been

possible owing to the patient's mental and physical condition, which has grown rapidly worse. (Seen in consultation with Dr. Wm. E. Fay.)

A study of these cases discloses several facts worthy of mention. In all four cases either an autopsy, an *x*-ray or physical signs showed involvement of the lungs. With the lungs involved, it is easy to understand how cancer emboli can be taken up by the pulmonary vein, carried to the heart and thence through the arteries to the meninges, choroid and other parts of the uveal tract. The lungs become involved either by direct extension, as from a mammary carcinoma, or through the lymphatics and the pulmonary arteries. The presence of cancer emboli in the arterial circulation is well illustrated by the case of Ishihara,¹ who described a primary mammary carcinoma with multiple carcinomatous emboli in the choroidal capillaries. There were three isolated tumor nodules in the choroid and eleven distinct capillary emboli composed of carcinoma cells. In one of Weeks'² cases an eye with a choroidal tumor was removed and the growth found to be carcinomatous with the primary source in the lungs only discovered at autopsy. A great many of the cases so thoroughly tabulated by Suker and Grosvenor showed an involvement of lungs when the records indicated a thorough general post-mortem examination.

The author queries as to how often, if ever, a choroidal carcinoma occurs until after some involvement of the lungs. The discovery, therefore, of a whitish discoid subretinal tumor with moderate or little elevation of the retina, no independent vascular system and no surrounding inflammatory reaction of the retina, should lead to a careful examination of the lungs, especially by the use of the *x*-ray.

In the author's first case, the tumor was diagnosed as a sarcoma. This has frequently been the case and, if the patient is first seen after the onset of pain and hypertension, is to be expected. Hypertension is not the rule, as in sarcoma, even when the eye is extensively involved. In my last three cases, including five eyes, hypertension was not present. How much the daily use of a miotic may have to do with preventing this painful complication can only be conjectured. In Case I pain and hypertension after arising were made to disappear by the use of cocain and miotics sufficiently to prevent any necessity of enucleation. Pain and hypertension are the only complications which, if unrelieved by treatment, call for an enucleation of the eye,

¹ Ishihara: *Klin. Monats. f. Augenh.*, liii, 127.

² Weeks: *Am. Ophth. Soc.*, 1915, xiv, 326. *Arch. Ophth.*, xlv, 554.

unless one is in doubt as to the differential diagnosis between metastatic carcinoma and sarcoma and, in such a case, the removal should only be advised after an exhaustive physical and x-ray examination has proved negative as to carcinoma. It is possible that the more extensive pushing forward of the vitreous due to the greater projection of sarcomata tends to increase the liability to hypertension while the flat carcinoma masses, even when extensive, do not encroach so much or so rapidly on the vitreous space and thus lessen the liability.

DIFFERENTIAL DIAGNOSIS

	<i>Metastatic Carcinoma</i>	<i>Sarcoma</i>
Situation.	Posteriorly, usually temporally and not far from the macula.	Usually equatorially, or farther forward, rarely near disc.
Shape.	Flat discoid with thin edges—rarely elevated more than 2 or 3 mm.	Rounded, nodulated.
Color.	Gray or white, without pigment.	Usually dark gray or showing much pigment and mottled.
Projection.	Very little if any projection into the vitreous.	Projecting far into the vitreous early.
Growth.	Spreads rapidly in all directions and ultimately surrounds the disc but always flat.	As size increases slowly projects more and more into vitreous.
New vessel formation.	Not present.	Usually present.
Retina.	Very slightly projected at first by the tumor growth and with little subretinal fluid—separation of the retina around the tumor comes quickly and spreads rapidly as the tumor grows and soon becomes complete.	Projected into the vitreous early by the tumor and separation of the retina around the tumor comes slowly and does not become complete until much later.
Vision.	Early disturbed and soon lost owing to rapid growth under or toward the macula.	Vision centrally not early disturbed and not quickly lost unless glaucoma appears—eyes with sarcoma often enucleated with vision normal.
Tension.	Not usually increased in early stage and usually amenable to miotics.	Usually raised early and acute glaucoma almost inevitable if eye not enucleated early enough—not affected favorably by miotics.
Enucleation.	Almost never necessary and of no value to the patient unless for otherwise unrelieved pain—does not prevent metastases.	Always necessary as early as possible and of great value to the patient in preventing metastases.
Occurrence.	Always metastatic and primary focus or other metastases can usually be found—often bilateral.	Always primary—seldom, if ever bilateral and causes metastases later.
Treatment.	Palliative.	Early enucleation.
Prognosis.	Inevitably bad.	Usually good, if seen early, though metastases of the liver may occur years later.

It is not possible to arouse much interest in such an absolutely hopeless condition, but one should be on the lookout for these cases, particularly as they seem to be on the increase. Finding an eye, therefore, that presents a discoid white, or grayish-white, subretinal mass located temporally not far from the macula, with the overlying retina only elevated 2 or 3 D. at the most and no evidence of inflammatory reaction, or independent vascular system, particularly in women, would be suspicious of metastatic carcinoma, remembering that such tumors rarely need enucleation.

In spite of an attempted differential diagnosis cases will occur later where an eye will be enucleated for sarcoma when the tumor is a metastatic carcinoma. In the author's first case this would have occurred but for the evidences of an enlargement of the liver. It is doubtful if the reverse is ever found, viz., that a tumor having, after careful study, been diagnosed as carcinoma, later turns out to have been sarcoma. This fact should be borne in mind when making a differential diagnosis. Therefore, when a tumor having all the characteristics described above as being indicative of carcinoma is so diagnosed and later the patient succumbs to what is very evidently a general carcinosis, especially if with evident lung and brain involvement, it may be safely considered that the correctness of a diagnosis of metastatic carcinoma of the choroid has been established, even if a microscopic study of the eye or a general postmortem be not made.

DISCUSSION

DR. GEORGE E. DESCHWEINITZ (Philadelphia): At Dr. Greenwood's request I exhibit a specimen of metastatic carcinoma of the choroid, the eyeball having been enucleated twenty-five years ago. The patient, a single woman, had carcinoma of the left breast, which was thoroughly removed. Six months later vision of the left eye became indistinct and examination revealed a flat carcinoma of the choroid. The patient died three months later, metastasis to the brain and lower end of the spine having occurred.

The tumor was composed of epithelial cells, rather spheroidal in shape, with large nuclei, collections (here and there) of small cells representing necrotic foci, pigment granules and a moderate amount of stroma. In some portions the carcinoma cells were arranged in long tubules, separated by faint stromal tissue, which contained pigment granules. A similar appearance has been described by Lagrange. In the region of the choriocapillaris, remnants of original choroidal structure were visible, also vessels plugged with cancer cells. The optic nerve and retina were normal; there was no change in the filtration angle.

A second case examined at about the same time concerned a married woman, aged forty-three, whose left breast was removed for carcinoma; re-

currence in loco, in the other breast and in the axilla occurred. The recurrent growths were excised; ten months later dimness of vision of the left eye was noted, and the typical appearances of metastatic carcinoma were found. The patient also had carcinoma nodules above the left clavicle. The patient was seen only once, and her subsequent history is unknown.

METASTATIC THYROID TUMOR IN THE ORBIT

DR. ARNOLD KNAPP

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Cohnheim was the first to recognize that a struma may cause metastases, and called them metastatic benign strumas. These metastases occur in the bones and in the lungs. Why they seem to select the bones is unsettled; A. Müller believes that the medulla of the bone furnishes a favorable site for their growth because of its retarded circulation. Thyroid gland cells are unusual in that they may, after entering the circulation, remain viable, and in certain places proliferate and form tumors. Trauma plays here a definite rôle. Striking is the destructive power of these metastases, which is particularly evident in the bones. This destructive tendency, the formation of metastases, and recurrences, cause some authors to regard them as essentially malignant. The thyroid in some of these cases seemed perfectly normal. The tumor may, of course, be overlooked in the thyroid gland.

Schmidt (*Zur Kasuistik und Statistik der Knochentumoren mit Schilddrüsenbau*, Inaug. Diss., Rostock, 1906) has collected 49 cases of struma metastases. In the metastasis, careful search reveals a carcinomatous part in the normal thyroid gland tissue. The site of these metastatic thyroid gland tumors is in various bones, particularly the cranial bones. Twenty-nine metastases occurred in the skull, principally in the frontal and parietal bones; in the vertebræ, 16; in the ribs and shoulders, 4; in the pelvis, 11; in the femur, 7; in the humerus, 6. The size varies from that of a fist to an egg. The age of the patient is between 30 and 60. It affects women more frequently than men. Trauma was elicited in 11. From the standpoint of the histology of the tumor and the clinical course, the struma metastases were malignant in 39; uncertain in 5; possibly benign in 5.

The following is an instance of this form of tumor:

Case Report.—E. R. B., aged 66; Dec. 18, 1917: Has always been in good health until recently. Has complained of vertigo and comes on account of discomfort in reading. Vision with glasses, 20/20. Both eyes seem unduly prominent. The right upper lid droops and the right eye is distinctly more prominent

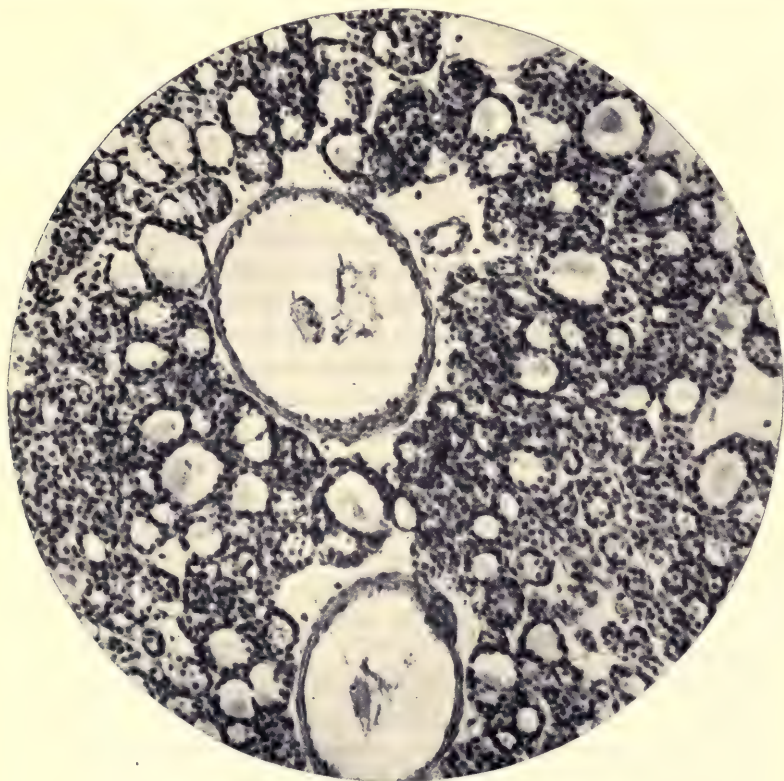


Fig. 1.—Metastatic thyroid tumor in the orbit.

than the left. Exophthalmometer—R. 32, L. 28. The motility of the right eye upward is restricted, particularly in abduction (superior rectus). Distinct vertical diplopia, increasing upward and to the right. This diplopia patient was observed for one week. On palpating the right upper orbital margin there is a resistance to be felt, especially in the region of the pulley, which consists of a soft mass within the upper margin of orbit, occupying a round defect in the bone, where pulsation can be felt. The pulley is displaced. Optic nerve nor-

mal. Field normal. The blood count is normal; hemoglobin, 90 per cent. The Wassermann test is negative.

The Roentgen examination shows an area of increased radiability on the right side, indicating an area of softening. It involves the orbital plate of the frontal bone and extends above the superciliary ridge. It is about three-quarters of an inch in its widest diameter. It extends about one inch above the supra-orbital ridge and along the orbital plate to the sphenoid fissure. There seems to be no involvement of the frontal sinus, but there is a supra-orbital extension of the ethmoid cells which seems to be very near indeed to this area of softening. With the exception mentioned, we find no indication of disease of any of the accessory nasal sinuses. The skull is unusually thick, especially the outer table of the frontal bone (H. M. Imboden).

Increasing doses of potassium iodid are prescribed for six weeks without any change in the tumor. An operation is advised.

Feb. 19, 1918, operation. Curved incision below the eyebrow down to the periosteum, which was found continuous downward with a mass. Incision also through the periosteum and the attempt made to elevate it. It was so firmly adherent to the underlying structure that this failed. The center of the mass seemed to be soft. The incision through the periosteum was then enlarged and immediately a dark, hemorrhagic-like mass resembling granulation tissue presented. The area was fully exposed, some of the periosteum removed and the above-described material was scooped out. The cavity was found in the bone extending back, upward anteriorly and laterally. Profuse bleeding made it difficult to see, and one had to be guided by a sense of touch. After cleaning out all this soft material and some of the rough bone along the margins, a rather well-defined cavity was exposed. The constant oozing was somewhat controlled by packing; it could then be seen that in two small places in the upper wall the dura was exposed without being directly involved. The edges of the bony cavity were trimmed off and the entire cavity packed with iodoform gauze. The external wound was left open. Length of operation, one hour.

In brief, this seemed to be a tumor arising in the medulla of the bone, particularly in the anterior part of the frontal, where it forms the upper wall of the orbit. The cavity was filled with soft, dark-red material. This in the lower part was directly adherent to the periosteum. A striking feature was the extensive bleeding, which seemed to be general. The bony walls of the cavity were smooth.

No reaction followed the operation. At the first dressing some of the packing was removed; considerable oozing.

The specimen removed at operation was sent to Professor James Ewing, who reported as follows:

"The tumor (Fig. 1) of the bone in the case of E. R. B. proves to be an adenoma of aberrant thyroid tissue. It is rather orderly in structure and not

very malignant, although in some spots the alveoli are small and numerous. It reproduces thyroid structure to the smallest detail, many alveoli containing soft acidophile colloid surrounded by flat thyroid cells. Many small alveoli are exactly similar to the usual thyroid adenoma. The stroma is scanty and not vascular.

"This tumor may arise from a portion of thyroid tissue originally present at that point in the embryo, or it may represent a metastasis of an adenoma in the thyroid. The thyroid gland should be examined for the presence of any small tumor at any point. I am inclined to prefer the former hypothesis, especially if there is no tumor found in the thyroid.

"The prognosis of these cases is not entirely favorable. Although they have been called 'benign metastasizing struma' they are not always benign. They recur locally, and the only other case in the skull which I have seen (Jeffries' case, in parietal bone) recurred locally and eventually produced metastases elsewhere. The thyroid was normal. Hence I recommend that radium be inserted in the wound, as the alveoli penetrate the bone spaces and are hard to reach by the knife. I know of no cases treated by radium, but would expect this structure to respond well (J. Ewing)."

February 24th: All of the packing is removed and a radium tube 27 mc. protected by a lead plate is introduced for four hours. The thyroid gland seems normal.

March 1st: No reaction, slight secretion superficially; wound is allowed to close.

April 1, 1918: The wound healed. A swelling remained at the upper margin of the orbit continuous with a bony mass externally just above the external canthus. Some exophthalmos remains, measuring R. 30, L. 28. Vision is normal. Eyeground normal.

November 26, 1918: General condition good; no change in orbit.

April 8, 1919: Has lost about 30 pounds in weight; complains of band over head and obscure abdominal symptoms.

July 1st: R. 31.5, L. 28. Diffuse swelling in orbit. Vision and eyeground normal.

October 8th: Drawing sensation in right half of head. R. 32, L. 28.

December 19th: The orbital condition is unchanged. Diplopia to the right. Distinct soft pulsating mass in orbit. A swelling had been noted over right scapula for some months and patient complains of neuralgia in right groin.

December 29, 1919: The patient is referred to Professor James Ewing, who reports as follows:

"There is a tumor mass behind the eye which causes distinct exophthalmos and protrusion of the supraorbital tissues of about 1 cm. The body of the right scapula is largely replaced by a tumor mass about 5 cm. in diameter, as shown by the x-ray. This tumor is of recent discovery and evidently growing actively. The left lower portion of the thyroid gland is the seat of a well-

circumscribed, rather firm tumor mass about 4 cm. in diameter. There is pain in the use of the right thigh muscles, which was not investigated, but will receive attention later. The *x-ray* of the right lung shows several suspicious isolated nodules, which I suspect are tumor nodules, but which cannot be positively identified as such. Further *x-ray* photos of the lungs and bones will be taken. The patient has lost weight, is anemic and rather feeble, and is therefore distinctly cachectic. I feel that the prognosis is unfavorable, but that some help may come from *x-ray* and radium treatment.

"I would recommend that the tumor of the scapula be treated by *x-ray* that the orbital growth be treated at first by a radium pack, and that the thyroid tumor be treated by the insertion of radium needles. This latter tumor is probably the source of the others. The scapular growth was treated this afternoon. We propose to go after the others slowly, but steadily, avoiding undue disturbance of the patient (J. Ewing)."

The shoulder tumor was given eleven *x-ray* treatments from December 29, 1919, to May 18, 1920. A radiograph on January 7, 1920, showed a destructive process in the eighth rib posteriorly and an area of bone destruction in pubis to right of symphysis. On January 20, 1920, a small area of destruction was found in the sixth rib posteriorly. The right groin was treated by *x-ray* four times from January 6 to May 4, 1920. Radiograph on January 7, 1920, showed dense shadow above manubrium and to the left of about the size of a small orange, indicating the presence of a calcified mass in the thyroid gland; pushing the trachea over to the right.

The orbit was treated with radium three times from January 19, 1921, to April 27, 1921.

The patient, according to Dr. R. W. Lowe, Ridgefield, Conn., then gradually lost weight, suffered from hallucinations and delusions, and was at times mildly maniacal. There was flatness over anterior and posterior chest (left); pleuritic pain, loss of motion over chest, dyspnea, slight cough. Lymphatic enlargement in neck (left side). Edema of lower extremities, increasing during last two weeks. Died July 21, 1921, apparently from cerebral hemorrhage.

In this patient a tumor in the roof of the orbit was the first symptom of a malignant process. At operation destruction of bone by a brownish, granulating, tissue-like mass was found present and unusually free hemorrhage occurred. The histology of the tumor showed it to be composed of thyroid gland tissue. No tumor could be detected in the thyroid gland on palpation. Symptoms of other metastases appeared one and a half years later and were confirmed by the *x-ray* examination in the scapula, in the sixth and eighth ribs, in the lungs and in the pubis, and at that time a distinct tumor was discovered in

the thyroid gland, and the x-ray showed that it extended behind the sternum.

The localization of these thyroid gland tumors in the walls of the orbit is unusual. In the literature there is a report of a case by von Eiselsberg (Langenbeck's Archiv, vol. xlv, p. 440) of a woman thirty-seven years old, who showed externally a bulging nodule in the substance of the parietal bone, projecting inward and adherent to dura. Another nodule was situated in the right upper orbital wall extending through into the skull. The sixth rib right and the left humerus were also involved; struma present (adenocarcinoma); autopsy report.

Jaboulay's patient (Bull. soc. de chirurgie de Lyon, 1903), a female, sixty-five years old, presented a tumor in the supero-internal angle of the left orbit, pulsating; old swelling of the thyroid gland. At operation the bone was found perforated, exposing the meninges.

In the discussion of von Eiselsberg's report (Verh. d. d. G. f. Chir., 1893, p. 88, i) Kraske said that he had observed a case where a tumor occurred in the frontal bone, the patient also having a struma. The association at first was not clear. At operation an unusual hemorrhage occurred, which is characteristic for this tumor. The tumor was adherent to the dura, necessitating excision of a part of this structure. The struma has remained stationary and no other metastases appeared. Microscopically the tumor resembled thyroid gland tissue and was regarded as an adenoma.

According to von Eiselsberg (*ibid.*, p. 255, ii), though the histologic structure of the tumor suggested adenoma, the clinical picture is that of an adenocarcinoma, because whenever a tumor metastasizes, it becomes malignant.

The metastases of the adenocarcinoma of the thyroid may show normal thyroid adenomatous tissue. The metastases grow slowly, while the primary tumor in the thyroid is small and escapes detection. The bone metastases should be operated upon, von Eiselsberg believes, though it is a general rule in surgery not to operate on bone metastases.

The primary tumor in the thyroid is often not found until the character of the metastases is made known.

BILATERAL LYMPHOSARCOMA OF THE ORBIT WITH INTERMITTENT EXOPHTHALMOS

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The subject of orbital tumors is an ever-increasing one which comprises a large field. The case at hand has several unusual features which warrant its presentation.

Mrs. M. B——, aged thirty-three, was first seen in 1908. Her symptoms were those of a refractive error, namely, hyperopia. The eyes were prominent without any apparent etiology. There was no thyroid disease and all other physical findings were negative.

August, 1917 (eight years later), patient returned, complaining of a slight drooping of the left upper lid. She was also under the care of Dr. A. W. Hewlett, Professor of Medicine at Stanford Medical School, who was treating her for an acquired lues. The eye findings were as follows: Slight ptosis of left upper lid. No apparent paralysis. Lid could be voluntarily elevated. Right globe somewhat more prominent than left. (At this time we did not possess an exophthalmometer.) Movements of eyeballs normal. Tension of left eye 32 mm., right 24 mm. No pupillary changes. Fundi negative. Perimeter fields, stereoscopic vision, and color sense all normal.

The following is an excerpt of the salient points in Dr. Hewlett's report:

"The general physical examination in October, 1916, showed the presence of numerous crackling and musical râles in the left back and axilla. Wassermann reaction was 3+. Sputum negative for tubercle bacilli. On March 3, 1917, the white blood-corpuscle count was 7,400, with 83% polymorphonuclear cells, 6% lymphocytes, and 11% large mononuclears. From March, 1917, to September, 1917, the patient was receiving anti-luetic treatment."

By October of the same year the ptosis of the left eye had increased, with a beginning exophthalmos of 3 mm. The patient continued under most vigorous anti-luetic treatment. November 21, 1917, the left eyeball became prominent. There was a marked swelling of the lids with chemosis. The

examination for leukemia was negative, the white-cell count being 7,500, with 81% polymorphonuclears, 6% lymphocytes, and 11% large mononuclears.

December 20, 1917, the right eye was beginning to show exophthalmos, while the left eye was at its height and with its upward motion limited. Eight days later the left was receding, and by January 15, 1918, both eyes were somewhat improved. January 20 the left eye was beginning to show recurrent exophthalmos, while the right continued receding, and on February 20 the left was at its acme for the second time, with the right normal. On March 1, 1918, the right was again markedly exophthalmic, with the left stationary, and by March 10 the left receded, leaving the right still somewhat swollen.

When the patient reported on April 5, 1918, the exophthalmos of the left eye was beginning to recur for the third time and the right was normal. On May 10 the left eye protruded markedly and the right was beginning to be prominent for the third time. As there was no improvement by July 6, 1918, the patient was put to bed, and by October 2, 1918, both eyes had receded; the therapy was limited to hot compresses and rest in bed. The left fundus now for the time showed a beginning optic neuritis.

A month later a circumscribed nodular swelling was observed in the bulbar conjunctiva at the nasal side of the left eye. There being a definite left-sided optic neuritis, the patient was sent to the University Hospital. A small vesicle developed which was punctured and showed a pure culture of staphylococci, the bacteriologic diagnosis being made by Dr. Karl Meyer.

The family history was negative. Father died at 81 (old age and asthma). Mother living and well. No Tb, malignancy, or other familial diseases.

Past history negative except for the usual exanthemata of childhood.

On January 20, 1919, the right eye was still normal, but the left showed for the fourth time a marked exophthalmos, and the patient was again sent to the hospital for further detailed examination.

Physical examination at this time was entirely negative. The blood count showed hemoglobin 75%; white blood cells 9,200; polymorphonuclears 70%; lymphocytes 26%; large mononuclears 4%; urine examination negative; stereoscopic x-ray examination of the skull negative; examination of nose: no evidence of sinus involvement; no involvement of preauricular or cervical glands.

EXAMINATION OF EYES.—Right eye normal. Left eye, marked edema of both upper and lower lids, with chemotic conjunctiva bulging through palpebral aperture. The globe protruded markedly, the exophthalmos being 10 mm. Entire conjunctiva injected and chemotic; at the inner canthus was a rounded nodule about the size of a pea, which appeared less edematous than the remainder of the swelling. The lower lid was overlapped by the growth while the upper lid was enlarged, covering the superior third of the mass, as shown in the illustration (Fig. 1). The eye was amaurotic and showed a secondary atrophy of the nerve-head. Under cocain anesthesia a small portion of the nodule was excised for microscopic examination.

The pathologic report of Dr. G. Y. Rusk, University of California pathologist, follows:

Specimen consists of a small, irregular mass of tumor removed from the subconjunctival tissue overlying the left eyeball.

Microscopic examination shows the tissue to be composed of a fairly uniform small cell, approaching a lymphocyte in size, but slightly larger and showing less condensation of the chromatic material. A slight rim of protoplasm can be made out in many of the cells. Mitotic division is observed with fair frequency, averaging about one to a high power field. Thin-walled vessels occur and also some with fairly well-developed walls. Extending from the vessels, fibers appear to run for a short distance between the cells. This ap-



Fig. 1.—Extent of exophthalmos on lateral view.

pearance, seen in the hematoxylin and eosin stained tissue, was confirmed by the use of the anilin-blue connective-tissue method. Away from the vessels no fine interstitial reticulum is demonstrable. In different areas the density of the cells varies, there being apparently some edema in places.

DIAGNOSIS.—Sarcoma of small round-cell type, possibly lymphosarcoma.

Later a diagnosis of lymphosarcoma was made and confirmed by Drs. Whipple and Meyer, of the University Staff, and by Dr. Ophüls, of the Leland Stanford, Jr., University, Department of Pathology.

January 20, 1919, the left eye was enucleated. It was debated whether to eviscerate the orbit or do a simple enucleation. We decided to remove as much tissue as seemed feasible, depending upon our operative findings. A circular incision was made just outside the limbus through the conjunctiva,

which was decidedly indurated. The ocular conjunctiva was dissected back, and the eyeball freed to the apex of the orbit. Muscles and nerve were then severed. The internal rectus muscle was obscured by a soft cellular tumor mass which involved it throughout its length. Digital palpation of the orbit showed the tumor mass to extend to the apex of the orbit and impinge upon the superior orbital fissure. There was no apparent bony involvement at the superior nasal border of the orbit. Due to the extensiveness of the growth and the likelihood of involvement of the other eye, as indicated by the tumor, and the practical impossibility of radical excision, evisceration of the orbit was decided as contraindicated. All available parts of the tumor were removed and the wound packed.

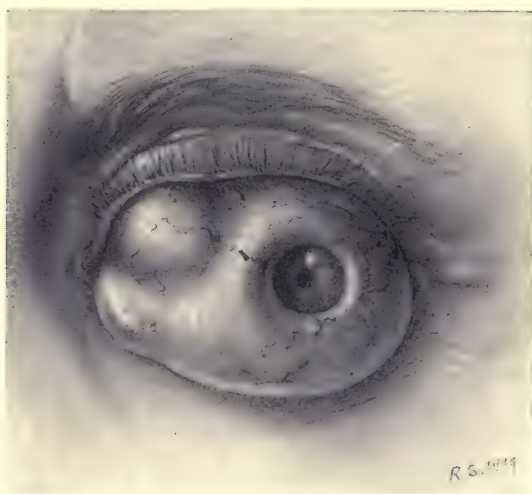


Fig. 2.—Anterior view with nodule at inner canthus.

The pathologic report follows: The eyeball and tumor in the case already reported (S. 19.9) was received in formalin. The tumor mass was loosely adherent to the ball except at the edge of the cornea, where it apparently merged with a circular opaque ring, completely encircling the cornea. The tumor measures $3\frac{1}{2} \times 2 \times 2$ cm. The eyeball and tumor were cut in the horizontal plane just above the optic nerve posteriorly and slightly above the center of the cornea anteriorly. From the larger portion of the bulb and tumor a block of tissue through the whole mass is taken and prepared for microscopic examination.

The details of the tumor correspond to those already described in report S. 19.9. One, however, gets relationships in a much more satisfactory manner. Anteriorly the tumor starts from the edge of the cornea and is covered

for some distance by conjunctival epithelium. It shows apparently little tendency to infiltrate the sclera, and in this region there is some edema of the pre-existing tissues. The tumor as a whole is somewhat lobulated, and while the main mass tends to grow away from the bulb, yet between the main mass and the bulb there is considerable tumor infiltration into the loose cellular tissue present. Toward the posterior end of the tumor extensive infiltration of muscle is observed. Connective-tissue septa of some size are observed in the posterior portion of the tumor, probably representing pre-existing struc-

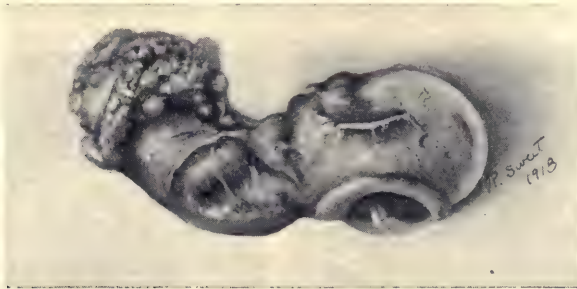


Fig. 3.—Gross specimen.

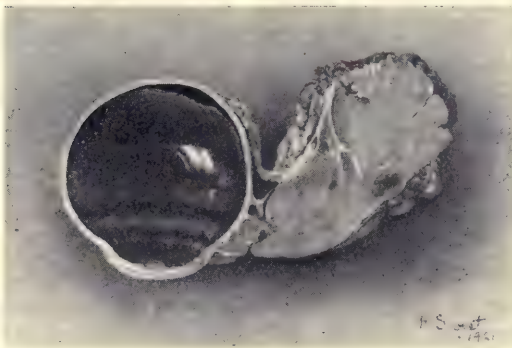


Fig. 4.—Cross-section showing involvement of internal rectus muscle.

tures, and these show varying degrees of infiltration. At the angle of the cornea and sclera on the side opposite to the growth there is some edema, but the growth has not encircled the cornea, as was suggested by the gross appearance. As to the eyeball proper, the retina appears normal, the pigment tissues in both the retina and sclera are normal, and there is no evidence of tumor having originated within the orbit. The optic nerve contains a suspicious number of clustered cells in places, small and round, which morphologically appear something like the tumor cells. Adjacent to the nerve, however, in

the angle which it makes with the sclera and on the side opposite the main tumor growth is considerable invasion of areolar connective tissue with loosely scattered masses of small cells, simulating strongly the tumor type.

DIAGNOSIS.—Lymphosarcoma.

On January 25, 1919, the patient received 350 milligram hours of radium to the left orbit (screened by $\frac{1}{2}$ mm. of silver and 1 mm. of brass) and an additional 300 milligram hours on February 3, 1919. Healing took place without signs of recurrence, although there was considerable skin reaction from the radium.

April 2, 1919, the right eye was negative to inspection and to all tests. The

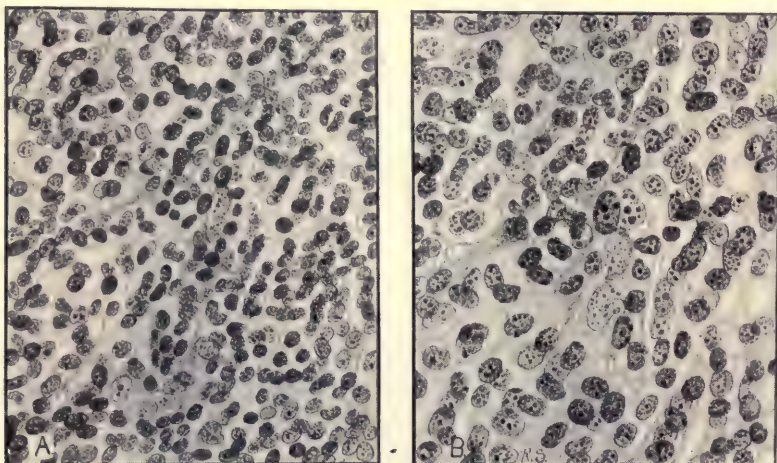


Fig. 5.—A. ($\times 581$.) Area with connective-tissue septum, showing lymphoid character of cells. B. ($\times 1000$.) Proportionate same area under high magnification. Illustrations show one-tenth reduction of original drawing.

left orbit showed a small palpebral aperture, slight symblepharon; no visible signs of recurrence. The patient was apparently well physically.

Up to and including April 26, 1921, the patient reported every few months for observation, and during that time there was no sign of recurrence on the left side, and the right eye was normal. The examination on April 26, 1921, showed V. R. E. 1/2. There was no exophthalmos, the exophthalmometer reading being 24 mm. The fundus was negative and the perimetric field for white and colors was normal. In July, 1921, we were informed that the patient had died, death being caused by an acute cardiac decompensation. There was an enlarged spleen, extending down to the pelvis, accompanied by a marked ascites. Several months previous patient had complained of a chronic cough, and on one x-ray examination a diagnosis of sarcoma of the lung was made. On a "retake" the diagnosis was altered to that of pulmon-

ary lues. Unfortunately, because of the religious creed of the family, it was impossible to obtain a postmortem examination. Clinically the entire picture was rather suspicious of a sarcomatosis.

COMMENT

The right eye of the case under discussion was presumably also effected by lymphosarcoma as its actions were identical to the left, with the exception of the appearance of the nodular mass. The case can justifiably be considered one of bilateral lymphosarcoma. Several unusual points are to be noted.

First, the bilateral occurrence of lymphosarcoma, with marked exophthalmos. The occurrence of lymphosarcoma of the orbit is rather infrequent. Chauvel¹ reports a single-sided lymphosarcoma, as does Würdemann,² while Meller³ describes a bilateral lymphosarcoma. Retrogression followed by progression was not noted in any of the above orbital tumors. Because of the infrequency of lymphosarcoma in the orbit, we had the pathologic diagnosis confirmed by several independent pathologists.

Bilateral exophthalmos due to retrobulbar conditions is comparatively rare. It generally occurs accompanying exophthalmic goiter and orbital varices. Syphilis had to be considered in this case, due to the triple plus Wassermann. In Forster's⁴ case of symmetric tumors with exophthalmos the condition subsided under KI. Bailiart⁵ reports bilateral symmetric gummata of the orbit. Anti-luetic treatment had no effect on our case.

Among the other causes of bilateral exophthalmos may be mentioned serous tenonitis (Tersen, A., and Tersen, J.⁶) and adenoids (Posey⁷).

Infrequent conditions to be considered are the various orbital tumors. Bilateral lymphomata are comparatively frequent. Becker and Arnold⁸ reported a case of bilateral lymphomata with exophthalmos and orbital glands which healed through extirpation. Gerlach's⁹ case, a four-and-a-half-year-old boy, showed lymphomata of the neck, liver, breasts, and conjunctiva. There were double-sided tumors of the orbital glands. Autopsy showed a generalized lymphatic involvement. There was symmetric exophthalmos of two years' duration, which extirpation showed to be due to lymphoma, in a case recorded by Bernheimer.¹⁰ Guaita's¹¹ patients showed diffuse lymphomata of the conjunctiva, as did Boerma's.¹² Arnold believes these to be due to hypertrophy of small lymphatic groups of

tissue that occur in the orbit. Goldzieher¹⁴ thinks they are due to follicular tissue from the conjunctiva that has grown into the orbit while Axenfeld¹⁵ considers them as misplaced fetal lymphatic tissue.

A large number of these tumors are found in cases of leukemia and pseudoleukemia. Leber¹⁶ reports such a case of double-sided retinal hemorrhage, together with numerous orbital tumors in a case of leukemia. Similar cases are also reported by E. Treacher Collins,¹⁷ Ostenwald,¹⁸ Deleus,¹⁹ Gayet, M.,²⁰ Raymond,²¹ and others. Ahlstrom²² goes so far as to claim that double-sided orbital tumors are always due to leukemia or pseudoleukemia. In his case of pseudoleukemia the orbital tumors were the first sign of the disease.

We considered the possibility of lymphomata, but all examinations for leukemia or pseudoleukemia were negative.

Among bilateral orbital tumors is a case reported by Henry Juler²³ of bilateral, round, and spindle-cell sarcoma, while Major Kirkpatrick's²⁴ patient had bilateral endothelioma of the sarcomatous type. In Silcock's²⁵ case, a girl of nine, the bilateral orbital tumors were round, all sarcomata.

Exophthalmos of the intermittent type due to circulatory changes in the orbit such as telangiectases, cavernomas, angiomas, and orbital varices, have been reported by Birch-Hirschfeld and Romeick,²⁶ Saint-Martin,²⁷ Hegner,²⁸ Enroth,²⁹ Alger,³⁰ Dupuy-Dutemps and Marvas,³¹ Posey,³² Mathus and Cerise,³³ and others. In none of the cases of intermittent exophthalmos was it due to an orbital tumor, although Birch-Hirschfeld³⁴ describes an intermittent increase of exophthalmos due to propagation of tumor cells in the orbital veins.

Another interesting feature in our case was the continued observation, post-operatively for a period of almost two and a half years, with no signs of recurrence on the operated side or the unoperated eye. It has been observed a number of times that removal of a tumor on one side has been followed by improvement of the unoperated eye. Among the more recent reports, Wilder³⁵ and also Lustig³⁶ made the same observation. The question arises as to the effect on the unoperated eye of the massive doses of radium and whether or not the effect was sufficient to check the tumor's progression.

We have been unable to find in the literature a case of bilateral lymphosarcoma with intermittent exophthalmos of the type described.

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CHLOROMA

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About one hundred years ago the first description of chloroma appeared in literature. Since that time cases have been reported from England, France, Russia, Germany, Austria, Italy, Japan, and the United States. Therefore it seems fitting that we should devote a few minutes to a disease that is even yet subject to discussion, not only from its etiologic standpoint, but especially in relation to earlier diagnosis and subsequent treatment, of which we know so little.

Four cases are here summarized, two of which have never been published:

CASE 1.—J. L., a boy eight years old, of foreign birth, entered the Albany Hospital December 3, 1906. The patient, semi-comatose, was extremely emaciated, and his skin was yellowish. The right eye was proptosed 11 mm., with a growth in the upper part of the orbit. The lower orbital region was outlined, but a freely movable mass, 12 mm. wide, extended from the outer to the inner canthus. This mass was not attached to either skin or periosteum, and its posterior margin was not palpable. There were two subconjunctival hemorrhages. Complete ophthalmoplegia externa. The cornea was clear. There was a marked optic neuritis, with complete obliteration of disc outline, with areas of white exudate along the larger vessels. Vision equal to fingers at six feet.

The left eye was extremely prominent, extending 20 mm. beyond the orbital ridge, with complete ophthalmoplegia externa. The veins surrounding the lids were enormously distended and tortuous. Projecting beyond the superior orbital margin was a firm, freely movable, not adherent growth, which extended deeply into the orbit and was palpable from the supra-orbital foramen outward. The conjunctiva was markedly chemotic, with numerous ecchymoses. The upper part of the cornea was clear, but the lower half showed a superficial necrosis and was covered with a dry exudate. Pupil 5.5 mm., not reacting to light or accommodation. There was a deep yellowish-gray reflex from the fundus. No detail; no light perception.

The right ear showed swelling of the posterior superior canal wall with tender mastoid. The left was almost the same. The lymphatic glands of the neck were palpable with difficulty on the right side, but on the left they were

visible as large as peas. Marked systolic thrill in the vessels of the neck. No abnormalities of the lungs.

Heart dullness began at the third rib and extended on the right side to the midsternal line. Apex-beat in the fifth intercostal space, 4 mm. inside nipple line. Loud systolic murmur over pulmonic area; second aortic sound accentuated. Apical sound clear and distinct. Pulse 120; tension good.

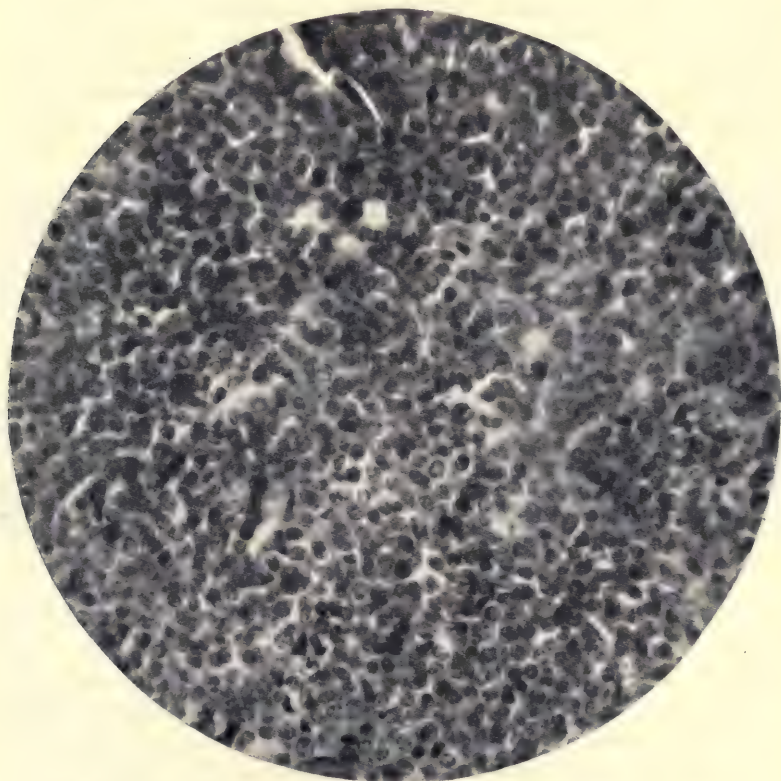


Fig. 1.—The larger type lymphoid cell in connective tissue. Case 1.

Moderate pressure on the sternum caused pain. Liver dullness extended from the sixth rib to the free costal margin. Spleen not palpable. Dullness from ninth to eleventh ribs. Abdomen negative except slight distention. Inguinal glands about the size of small peas. Umbilical, costal, and plantar reflexes normal. No knee-jerks.

Urine clear, amber, acid, s. g., 1020; no sugar, but a trace of albumin without casts.

Blood examination made by Dr. A. T. Laird showed: Red cell count, 1,410,000; white cell count, 79,600; hemoglobin, 30 per cent.; and a differential count of 6,000 leukocytes showed polynuclears, 16.2 per cent.; large mononuclears, 5.3 per cent.; large lymphocytes, 15.3 per cent.; transitionals, 0.8 per cent.; eosinophiles, 0.7 per cent.; small lymphocytes, 33 per cent.; neutrophile myelocytes, 28.2 per cent.; eosinophile myelocytes, 0.5 per cent.

Five nucleated red cells were noted: three were normoblasts, two were larger than normoblasts and were classed as megaloblasts. A number of degenerated leukocytes were seen, but no record of them was made.

The patient was given chloroform, and a 3 cm. incision was made over the outer half of the superior orbital ridge. The entire orbit was found filled with a greenish mass of almost cartilaginous consistence, in part encapsulated, nowhere palpably adherent to the periosteum. We could outline the optic nerve, but could not distinguish any of the ocular muscles. Palpation to the apex of the orbit proved that the entire contents consisted of a pale green tumor which was diagnosed as probably chloroma.

The tissue removed was sent to Dr. R. M. Pearce, of the Bender Laboratory, who reported as follows: Tissue from left orbit: Material consists of three small pieces of tissue, the largest about 0.5 cm. in diameter. These are irregular in shape; in part firm and of light greenish color, and in part soft and pink in color. On section they show a uniform, smooth surface, firm in the green but soft in the pink portions.

Histology.—The centers of the nodules are composed of closely arranged cells of the type of the larger lymphoid cell. These have a deeply but slightly irregular staining nucleus and a small ring of eosin-staining protoplasm. As a rule, they are round or slightly oval in shape, but occasionally are irregular, resembling in a general way the plasma cell. No multinucleated cells are seen. Toward the periphery are single fat spaces which have resisted the general infiltration. These spaces are more abundant at the extreme periphery, but the tissue between is extensively invaded by the new cells. Only here and there are small areas of uninvolved fat tissue seen. Here also are areas, apparently at point of periosteal attachment, with considerable fibrous tissue infiltrated with lymphoid cells and more or less hyaline in character. In the center of the nodules there is but a faint reticulum of indefinite arrangement. Few blood-vessels are seen; polymorphonuclear leukocytes are not present in appreciable numbers.

Histologic Diagnosis.—Chloroma.

Dr. James F. Rooney saw the patient November 26, 1906, when the blood showed: reds, 1,596,000; whites, 41,200; hemoglobin, 30 per cent.; color index, 1+. Differential count of 400 whites: polymorphonuclears, 37.9 per cent.; large mononuclears, 11 per cent.; small mononuclears, 12 per cent.; eosinophiles, 3.1 per cent.; myelocytes, neutrophilic, 32.8 per cent.; myelocytes, eosinophilic, 7.2 per cent.; 5 nucleated reds, all normoblasts; poikilocytosis, many microcytes and macrocytes; many degenerated whites.

The patient's condition grew progressively worse from the date of admission. Emaciation was extreme. Proptosis of both eyes more marked. On the right side the tumor mass increased 2 mm. in width and the eyelid became darkly congested, with tortuous, prominent vessels. Vision almost totally lost. Pupil 6 mm., not reacting. No increase in the retinal changes. The lower half of the cornea of the left eye was infiltrated, and the outer layers eroded. No fundus visible; no decrease in the chemosis; veins more enlarged. No drainage from the wound. The glands of the neck were greatly enlarged, on the left side being as large as pigeon-eggs. Veins of the neck showed no signs of thrombosis.

On December 10th the child had a convulsion, which started with general muscular tremor of the right side, followed by marked contraction with turn-



CASE 1.—The extreme exophthalmos of the left eye with a marked engorgement and discoloration of the eyelids.



CASE 2.—Bilateral exophthalmos.

ing toward the left. The left side was entirely paralyzed; respirations stertorous; patient unconscious. He rallied, and from time to time seemed to comprehend things. He was removed from the hospital and died that day. Autopsy was not permitted.

CASE 2.—Mrs. N. G., aged eighteen, married, a Russian by birth, was admitted to the Albany Hospital April 7, 1909. Family and personal history negative. Patient menstruated from thirteen years of age until one week after marriage, July, 1908. One month prior to admission she first noticed many small, hard, insensitive lumps in each breast, which she believed had not increased in size. March 8, 1909, she had "pink eye," from which she seemingly recovered; on March 27th her eyes began to bulge, causing pain and attacks of blindness.

The patient was an undersized, delicately built, poorly nourished woman.

The skin had a peculiar satiny texture and was of grayish-yellow color. Several nodules, about 2 cm. in diameter, which were attached to muscle and bone, were palpable in each breast and along the sternum. The axillary and inguinal glands were enlarged. The woman was eight months pregnant; a systolic murmur, audible over the whole precordium, was not transmitted.

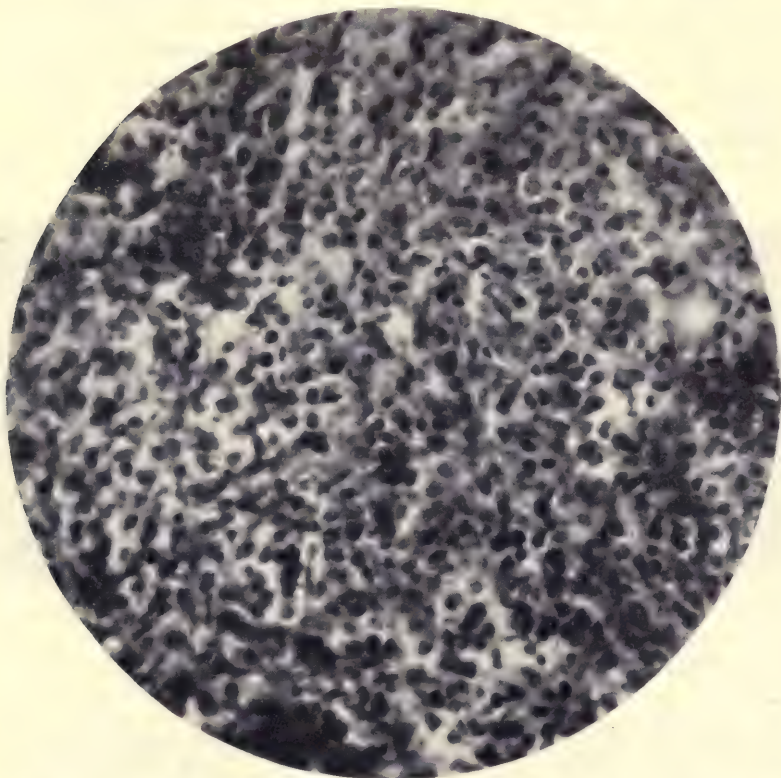


Fig. 2.—Closely packed large round or oval cells of the lymphatic series; cytoplasm variable in amount, also many cells having fine and coarse granules resembling myelocytes. Case 2. The section of Case 3 was practically the same, except that eosinophiles were found throughout.

These were the only departures from an otherwise negative physical examination, except for the ocular lesions and blood changes.

April 8th the right eye proptosed 20 mm.; immobile. The upper lid showed great dilatation of the blood-vessels, and although it covered one-third of the cornea, it could be retracted, while the lower lid was covered with chemotic conjunctiva. A definite tumor mass, 20 by 12 by 8 mm., was outlined over the lacrimal gland, entirely in the lid, with no deep attachment. The superior

two-thirds of the cornea was clear, but the lower third showed a dense interstitial and superficial haze. Pupil 1.5 mm. Vision = fingers at 3 feet.

Left eye proptosed 18 mm.; small nodule in the upper lid at the outer side; cornea hazy below; pupil 4.5 mm., stationary. Vision = fingers at 3 feet. A large mass of grayish appearance pressed the retina forward, and except for an occasional vessel, obscured fundus detail.

Blood examination made by Dr. James F. Rooney, April 8th showed: reds, 3,150,000; whites, 36,500; hemoglobin, 55.0 per cent. Differential count of 300 white cells: polynuclears, 15.8 per cent.; lymphoblasts, 37.2 per cent.; small mononuclears, 37.0 per cent.; large mononuclears, transitionals, 9.0 per cent.; eosinophiles, 0.8 per cent.; mast cells, 0.2 per cent.

Five normoblasts seen in counting 300 leukocytes.

Four days later, April 12th, the condition was as shown in the illustration. The right eye bulged forward 25 mm., with intense engorgement of the veins of the lids and temporal regions. The tumor was now 25 by 12.5 mm., axis 60 degrees, not adherent to the skin or periosteum, although extending deeply into the orbit. The outer part of the lower lid was filled by an oval, unattached growth, 18 by 9 mm., axis 120 degrees. The lower half of the eyeball, including the entire cornea, was exposed. The conjunctiva was chemotic, with many small ecchymoses, and a small hypopyon in the anterior chamber. Pupil 2 mm., stationary, tension normal; extremely limited motion of the globe; vision = light perception.

Left eye proptosed 22 mm., with many irregular nodular masses in the upper outer two-thirds of the orbit, close to the superior wall, but not adherent to it. The upper half of the cornea was covered by a vein-filled upper lid; the lower half was rough and infiltrated. The bulbar conjunctiva was decidedly congested and the eyeball stationary. The interior of the eye was as before. Pupil, 4.5 mm.; faint reaction to light; tension normal.

Dr. Rooney's blood examination, April 12th, showed: reds, 2,200,000; whites, 52,000. Differential count of 300 whites; lymphoblasts, 42.5 per cent.; polynuclears, 10.6 per cent.; small mononuclears, 19.2 per cent.; large mononuclears and transitionals, 8.0 per cent.; eosinophiles, 3.0 per cent.; myelocytes, 0.7 per cent.; degenerated, 1.0 per cent.; six normoblasts, one megaloblast.

The disease was diagnosed chloroma by the examination of breast tissue. This was done before the patient died, on April 16th, after premature delivery of a dead child. As in many cases, persistent, uncontrollable nasal hemorrhage was present for several days before death.

The following is the autopsy report of Dr. Wolbach, held April 16, 1909:

Autopsy limited to chest and abdomen, so that the orbits were not investigated.

Body is that of a short-statured, slightly built white woman. There is extreme double exophthalmos, and both eyeballs are reddened and covered with pruriform material and crusts. Breasts are large and nodular. The areolæ are deeply pigmented. Midline of the abdomen is deeply pigmented. Rigor mortis is complete. Marked postmortem lividity of dependent parts. No edema.

Peritoneal cavity: Peritoneum is smooth, moist, glistening. Appendix normal. Mesenteric lymph-nodes not enlarged.

Chest: Under surface of the sternum is covered with many green tumor nodules, which are situated on the surface and beneath the periosteum. The intercostal muscles on both sides for a distance of several centimeters are invaded and replaced by tumor tissue which completely surrounds the costal cartilages of the upper five or six ribs.

Pleural and pericardial cavities negative.

Heart: Normal in size. Myocardium is of good consistence. Valves and endocardium are normal. There are three tumor nodules in the auricles of the heart, situated as follows: one in the posterior wall of the left auricle, near the interauricular septum. This nodule is olive green in color and measures 2 by 1.5 cm. In the interauricular septum, just above the mitral valve, and close to the posterior border, is a nodule 0.5 cm. in diameter, which is pale green in color. In the wall of the right auricle, in the posterior side, close to the interauricular septum, is a similar nodule, 1 cm. in diameter.

Lungs: Both are negative, except for edema and congestion. At the root of the left lung, presumably in the mesenteric nodes, are two pale green tumor nodules about 1.5 cm. in diameter.

Spleen: Normal in color and consistence.

Liver: Pale, reddish brown in color, normal in size and consistence. No tumor nodule found on close inspection.

Pancreas: There is a mass of large, green glands at the head of the pancreas. On section, these vary in color from a pale green to an olive green. Embedded in the tail of the pancreas are two similarly colored nodules, about 1 cm. in diameter.

Gastro-intestinal Tract: Not opened.

Kidneys: Both kidneys are normal in size, pale in color, and each contain many nodules, varying in size from 2 mm. to 1.5 cm. The largest are situated beneath the capsule, and are flattened and soft in consistence. Smaller ones are distributed throughout from cortex to pyramids, and are firmer in consistence. These nodules are of rather pale color, but of a decided green tint. The larger ones show small red areas—presumably hemorrhages.

Adrenals: Both are normal.

Prevertebral lymph-nodes are markedly enlarged. On the left side, just

above the bifurcation of the aorta, is a large mass of pea-green tumor tissue which is firmly attached to the periosteum covering the vertebræ. This mass is roughly hemispheric in shape, with a base 5 cm. in diameter and a depth of 3 to 4 cm. Consistence is very firm.

Genitalia: Uterus large and has the partially contracted appearance of a recent delivery. Ovaries and tubes normal. Owing to the lack of time, genitalia were not more closely inspected.

Fresh microscopic examination of the tumor shows it to be made up of small round cells, a few of which contain many refractive granules. The fluid obtained by teasing and expressing glands contains many refractive globules, which on exposure to fumes of osmic acid turn dark brown.

Breasts: Both breasts and surrounding fat tissue are largely replaced by green tumor tissue. The growth consists of spheric and ovoid masses, from a size just visible to nodules 3 cm. in diameter. These nodules are of quite firm consistence. The color varies from a pale green to an olive green.

Anatomic diagnosis: Chloroma with metastases to sternum, heart, bronchial nodes, kidney, pancreas, and lymph-nodes.

CASE 3.—B. R., aged five years, entered the Albany Hospital April 23, 1913, and was discharged May 16, 1913. The dominant symptom was a firm, ovoid growth occupying the upper one-half of the right orbit, producing the effect, as shown in the illustration, of a mass anterior to the eyeball. The skin was freely movable over it, but the tumor extended deeply into the orbit. The right eye was movable in all directions, without fundus lesion. The left eye and orbit were uninvolved. The patient had a peculiar sallow appearance. He had several slight hemorrhages from nose.

The blood count showed: White blood cells, 7,700; red blood cells, 1,930,000; hemoglobin, 55 per cent. (Tallquist); differential white count, 250 cells; polymorphonuclear neutrophiles, 25.6 per cent.; small lymphocytes, 65.6 per cent.; large lymphocytes, 5.6 per cent.; large mononuclears, 1.6 per cent.; eosinophiles, 0.4 per cent.; transitionals, 1.2 per cent.; irregularity in size and shape of red cells; no nucleated red seen.

The day of his discharge showed: White cell count, 6,200; red cell count, 1,510,000; hemoglobin, 50 per cent. (Tallquist); differential count, 250; polymorphonuclear neutrophiles, 12.8 per cent.; small lymphocytes, 70.4 per cent.; large lymphocytes, 2.4 per cent.; large mononuclears, 14.4 per cent.; the red cells are somewhat irregular in shape and size. The increase in mononuclears is noteworthy.

The temperature ranged from 99° to 102° F. Wassermann reaction was negative. Before the child was taken home, where he died June 5, 1913, the right eye became very prominent, with practically no motion. Definite masses, similar to that in the upper lid, also appeared in the lower, filling the orbit. A firm, lobulated tumor deep in the left orbit produced marked exophthalmos. The temporal region became infiltrated. The hearing was greatly

reduced. There was no marked glandular enlargement. The patient died with meningitic symptoms.

On April 26, 1913, the patient was given an anesthetic and a curvilinear incision was made close to the superior orbital margin. The entire orbit was filled with a firm green mass. Part was excised for histologic study, and the diagnosis of chloroma confirmed.

During the time of the patient's stay in the hospital he was given benzol without improvement. Despite strenuous efforts, autopsy was not permitted.

CASE 4.—J. N., a girl seven years old, of foreign extraction, entered school a year ago, and seemed to be normal in every way. After being in school a few weeks she began to complain of headache and would cry for hours. On July 4, 1921, the child's right arm was severely burned and she was sick for three weeks. On October 27, 1921, Dr. LaSalle Archambault sent her to my



Case 3.—Ovoid mass in the right upper lid which extended far into the orbit.



Case 4.—Marked bilateral exophthalmos greater on the right side with temporal fullness.

office. The right eye bulged forward 11 mm., with limitation of motion vertically but not horizontally. There were several large, rounded masses freely movable beneath the skin, but extending into the orbit and seemingly adherent to the superior orbital ridge. There were also masses in the lower portion of the orbit, with the same characteristics. The pupil was 6 mm. stationary, media clear, with an intense neuroretinitis, many areas of whitish exudate and several flame-shaped hemorrhages. The left eye protruded 8 mm., with the same type masses extending deep in the orbit. There was definite swelling of the optic nerve, with retinal exudate. In both temporal regions there was a firm swelling and the patient was totally deaf.

I sent her to the Albany Hospital with a diagnosis of chloroma. This was verified by the blood examination, which showed: Red blood cells, 3,100,000; white blood cells, 20,800; hemoglobin, 67 per cent.; polymorphonuclears, 19

per cent.; large and small lymphocytes, 81 per cent.; large mononuclears, 68 per cent.; small mononuclears, 32 per cent.; benzidine reaction shows the lymphocytes to be of the myeloblastic series. The second count, October 30, 1921, showed: Polymorphonuclears, 21 per cent.; large lymphocytes, 6 per cent.; small lymphocytes, 12 per cent.; large mononuclears with granular cytoplasm (myelocytes and myeloblasts), 54 per cent.; transitionals, 7 per

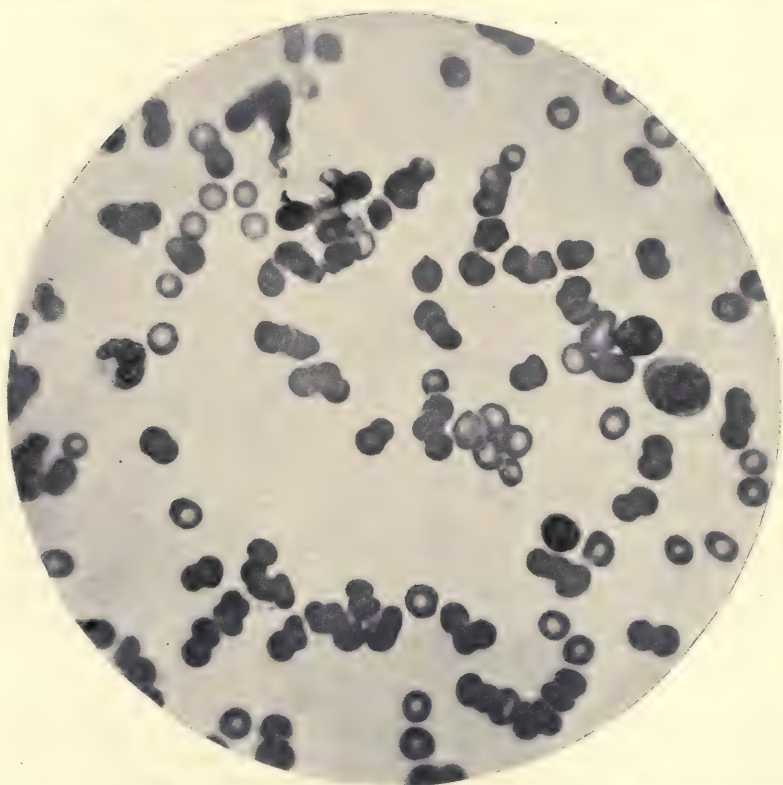


Fig. 3.—Blood smear from Case 4. Wright's stain, showing the usual red blood-cells, polymorphonuclears, lymphocytes, and myelocytes.

cent. Oxydase stain showed granular forms in the ratio of 9 to 1 non-granular.

Urinalysis: Color, amber; cloudy; reaction acid; sp. gr. 1017; albumin positive; sugar negative; microscopic—loaded with amorphous urates, mucus.

Temperature, which was on admission 100.3°, dropped to 98° the next morning, but then rose above 103°. The parents took the child from the

hospital when they were told that the outcome would be fatal. The child died at home November 9, 1921, and it was impossible to get an autopsy.

In the complete review of the literature we find that 90 cases have been reported—25 females, 5 sex not stated, and the remainder males. The age varied from one year nine months to fifty-five years, by far

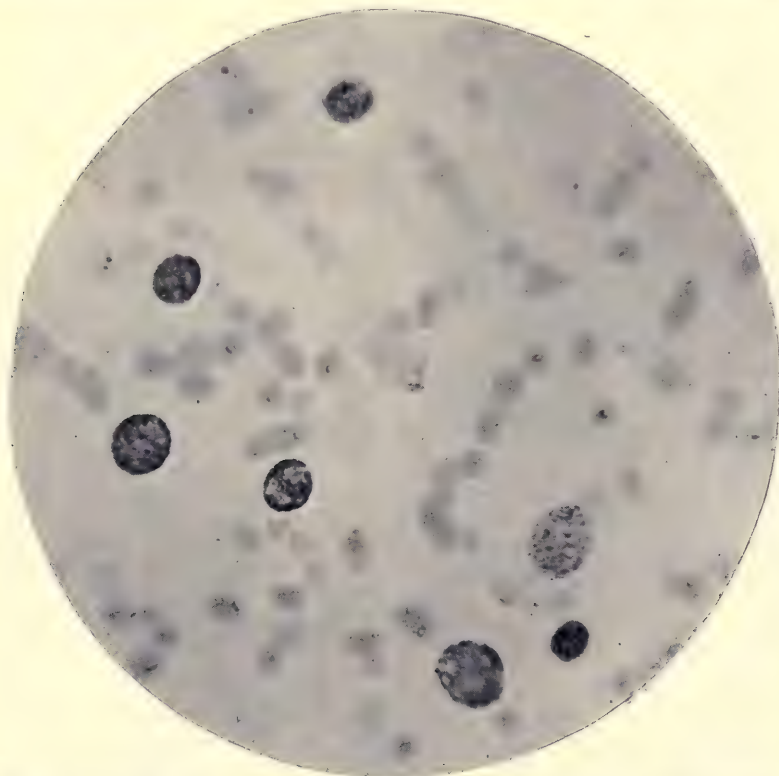


Fig. 4.—Oxydase reaction of the blood of Case 4, showing polymorphonuclears, myelocytes, myeloblasts, and nuclei of small lymphocytes.

the greater number being in children and young adults. The duration of the disease was so uncertain that even the statement that many died within one month, some even within four days after the primary visit, helps little in the diagnosis. The greatest number of cases were only properly understood at autopsy.

The reported white counts varied from 6,200 to 519,600. In only six cases, however, was it 100,000 or more. The commonest count is

less than 40,000, and, as will be noted in our own cases, one count was 6,200. On the other hand, the differential count gave information of the greatest value, for in all cases we find a marked decrease in the polymorphonuclears, with a considerable increase of the myelocytes.

The diagnosis of this condition can be made early. Every case of exophthalmos in a child should have an immediate complete blood examination, for, as has been proved by many and corroborated by our cases, the first symptom is in the blood change.

The treatment has ranged from benzol to α -ray to radium. The lack of curing power when late, at least, is evidenced by the fatal outcome of all cases.

To summarize: Chloroma, a disease of the blood-making organs, lends itself to early diagnosis by its characteristic blood picture. The common eye symptoms are lid and orbital tumors, exophthalmos, subconjunctival hemorrhages, retinal hemorrhages and exudate, neuroretinitis, and blindness, although frequently unilateral, most often the changes become bilateral and, finally, investigation will doubtless prove an infection the origin of the disease, and that such infection will be diagnosed and cured. It is with this hope that this clinical communication is presented.

It is with great pleasure that I acknowledge my indebtedness to Dr. C. S. Merrill, who so kindly made it possible for me to diagnose and treat Cases 1 and 3; to Dr. G. Emory Lochner, for giving me the control of Case 2; to Dr. Charles E. Allen for his assistance in Case 4, and to Mr. J. A. Glenn for the microphotographs of tumors and blood, and to my able and conscientious assistant, Dr. Anton S. Schneider.

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PROPOSITIONS FOR CONGRESS ACTION

THE VALUE OF LETTERS AND CHARACTERS AS VISUAL TESTS

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Since the fixing of the visual angle by Hooke (1674) and its practical adaptation by Snellen to test letters (1862), there have been efforts on the part of various investigators to find an object or objects of universal recognition that could be made use of to represent this angle for the accurate testing of vision. The ideal has not yet been attained. The nearest approach to its attainment has been the employment of letters from the alphabets of various languages. For the Anglo-Saxon, the Latin and the Teutonic peoples the block letter was suggested by Snellen, was used by him in his charts and has become the almost universal standard in these countries. Snellen realized that these letters offered only an approximately correct representation of the relative visual acuity, even though certain openings in the lines constructing the letters indicated the one-minute angle, as the opening in the letter E and in the letter C, the two representative letters among those used for measuring the vision, the one being round and the other square.

The realization that letters were variable and faulty in the attainment of uniform visual results led to the suggestion of the so-called one-minute tests, as the Landolt "broken ring," the "interrupted square" of Jackson, the central dot of Wolffberg and the broken line test.

The difference in the values of various letters as visual tests was pointed out by Green in 1872, Dennett in 1885, Randall in 1894, and later by Jackson. When Snellen first adopted the "block" or "Egyptian paragon" letters he recognized these differences and for this reason he selected letters which would approach as nearly as possible to his three line, one and five minute standard square.

In order to get at this subject more definitely, at the suggestion of Dr. Jackson a committee was appointed by the Ophthalmic Section of the American Medical Association in 1914 on "Standardizing Test Cards." At the further suggestion of Dr. Jackson, the members of this Committee selected all the appropriate square and round letters in the alphabet, both in the "Egyptian paragon" or "block" form and in the "Gothic" or "antique" form, the two forms in general use, and working separately with various patients of all types, each committee-man gave his conclusions as to the visual value of each of the letters as compared with the Landolt "broken ring." The average percentage visual value of these results is shown in the accompanying table.

<i>Gothic</i>		<i>Block</i>	
L.....0.62	F.....0.81	L.....0.71	P.....0.81
A.....0.71	C.....0.85	T.....0.74	D.....0.82
T.....0.72	K.....0.88	V.....0.78	Z.....0.84
V.....0.74	O.....0.88	U.....0.79	N.....0.85
H.....0.74	D.....0.88	C.....0.79	E.....0.85
Z.....0.76	X.....0.91	O.....0.80	R.....0.88
E.....0.77	R.....0.93	Y.....0.80	S.....0.89
N.....0.79	S.....1.05	F.....0.81	C.....0.92
P.....0.79	B.....1.16		H.....0.92
			B.....1.00

These results revealed that few of the letters could be depended on to give so-called normal visual acuity as compared with the "broken ring" of Landolt. They further confirm Snellen's observation that the "block" letter was superior to the "Gothic" letter for the construction of visual test-types. Also they standardized so far as was possible the relative visual value of each letter.

In this table it is shown that the letter L is the most readily seen of any of the letters at a given distance, but it is well known to all ophthalmologists that persons who are able to recognize the L have sufficiently good vision to pursue any of the ordinary vocations necessary to the earning of a living. In this lies the reason why test letters are almost universally employed as visual tests. In other words, the Committee has demonstrated that persons with 0.62 per cent. of so-called standard or normal vision, see well enough for all ordinary purposes.

"In all times letters and numbers have been preferentially employed by ophthalmologists in the investigation of the power of vision," observed Donders in his work on refraction in 1864. After the lapse of another half century, during which period workers in

ophthalmology have increased by thousands, it may be safely claimed that this observation is now an axiom, with letters still in the ascendant. The investigations of the Committee above mentioned have given each letter a definite visual value in comparison with an accepted visual standard. Experience has proved that a visual acuity of 75 per cent. of this visual standard indicates sufficiently good vision for even the highest grades of occupations in securing a living. The army accepted 50 per cent. as sufficient for ordinary occupations, and practically 75 per cent. for the finest rifle work, as the vision of the riflemen was taken as a rule with Snellen type, and the work of the Committee has shown that the visibility of this type ranges all the way from 66 to 100.

Another circumstance that has been confirmed by experience is, that ophthalmologists as a rule consider the so-called "one-minute" tests as nuisances and time consumers, and will not bother to employ them when letters and characters serve the purpose. When great accuracy is required, or an exact comparison is necessary, it is well to have some one of these tests at hand.

With the above data at our command the rational method of disposing of this vexed question of visual acuity would be to accept, as the standard or normal vision, the vision measured by any of the block letters from T to H in the report of the Committee, when these letters are drawn to the one- and five-minute scale of Snellen. The same rule should apply to characters when they are drawn practically within these limits. This would mean a variation in the visibility of 18 points, but experience has further demonstrated that this counts for little after the 75 point limit has been passed. The reading of any one of these letters should be considered as the 100 mark, instead of the reading of the broken ring of Landolt, and visual acuity above this standard 100 mark should be indicated as normal plus. Such an arrangement would satisfy every visual requirement.

OPHTHALMOLOGIC VERSUS ANATOMIC NOMENCLATURE

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It is a rule in anatomic descriptions to consider the subject standing in front of the observer, who, when speaking of the right side, for instance, always refers to the subject's right, which is in front of the observer's left hand. This is a basic rule and all anatomic treatises in the medical literature of all countries conform to it.

The same rule holds good in physiology, in surgery (when not otherwise specified), in clinical methods of examination, etc. The description of one operation is easily understood when the author conforms to the anatomic nomenclature and the reader can visualize the different stages of the interference with the organs as they really are in front of him. In this way the necessary repetition of "patient's right or left side" or "surgeon's right or left" is avoided, and medical descriptions will gain in accuracy, clearness, and conciseness.

In ophthalmology, unfortunately, the official anatomic nomenclature is not followed up altogether, with the resultant misunderstanding, confusion, and unnecessary mental strain.

Diseases of the anterior segment of the eye are studied in the textbooks and treatises generally according to the anatomic standard because this is unavoidable, the illustrations necessarily showing the patient's eye facing the observer; but the confusion begins when the pathologic sections are represented inverted, the eye in the same position in space as the physician's eye. Passing from the description of the changes to the study of the sections, an effort of imagination is necessary to replace the organs and lesions in the proper position. In this way two different standards are set up and the relation of the parts and structures to each other changed, without sufficient reason or practical benefit.

In the description of the normal and pathologic iris, for instance, the membrane is represented outstretched in front of the observer, whereas the microscopic sections show the posterior or pigmentary

layer, forward. When the ophthalmologist is accustomed to remember the angle of the anterior chamber, in the non-anatomic position, it is very difficult for him to transpose it to the proper place if the new method of examination with the contact glass and the ophthalmoscope (ophthalmogonioscopy) is used. Many competent observers fail to grasp the relations of the ciliary body with the anterior chamber, its continuity with the sclera, the position of the sclerocorneal trabeculum and Schlemm canal in relation with the cornea, etc.

The different systems of blood-vessels in the anterior part of the eye, easily understood in the old schematic figures, become difficult to represent in their true position with the patient's eye in front, particularly the relations of the perforating ciliary vessels with the intra-scleral network and the iris and ciliary vessels.

In glaucoma, when there is an adhesion of the root of the iris to the cornea, it is easier to visualize the formation of the new iridocorneal angle and correlate it with the diminution in depth of the anterior chamber, the enlargement of the pupil and its irregular shape due to partial adhesion, if the mental picture of the structures has been made in the anatomic position.

The confusion and misunderstanding grow worse in the study of the optic nerve, optic tracts, decussation in the chiasm and the relations of the bundles of the nerve-fibers and tract forming a forked sensorial organ, with the two lateral halves of the retina and the visual field.

The well-known schematic figures of the optic pathway are always printed considering the observer standing at the back of the patient. This was probably made in order to facilitate the understanding of the visual fields which, as recorded by the campimeter and perimeter, are also inverted and shown in the non-anatomic position, as we will consider later.

The transfer and reversal of the visual field found in hemianopsia and the location of the homonymous halves of the retina to the proper side of the patient have been the cause of the great obscurity, misunderstanding, and mental effort which generally confronts the student in this particular subject. Homonymous is taken generally as meaning the two halves of the same name in the retina,—for instance, both nasal or temporal halves,—while in reality they refer to the patient's right or left side.

Any one who will study this subject according to the anatomic standard will readily realize the numerous advantages derived, and the facility to memorize the lesions.

It is, however, in the diagnosis of the troubles of motility of the eye where the principal difficulties arise for the student; an earnest effort and great mental strain are required to transpose the mental images from one position to the other. The anatomy of the ocular muscles is taught in reference to the observer's eye and he needs to memorize the insertion and action of the six extrinsic muscles in this position. Afterward in clinical examinations, with the patient in front of him, either watching the limitation of movements of the eyeball in case of paralysis of the muscles, or when moving the candle from one side to the other in the subjective examination for diplopia, he will constantly need to reverse his mental images or go back and place himself behind the patient to be able to arrive at a diagnosis.

The excellent Fuchs text-book, fifth English edition, page 713, shows, in Fig. 301, the place and insertion of the ocular muscles in the inverted position, and side by side the next figure represents the eye in the anatomic way. The four frozen sections of the orbit and the schematic plan of the action of the muscles after Marquez are seen from before, as are also the lines of insertion of the four recti, but a few pages later, in the chapter on paralysis, the inversion reappears, and although the student must detect the primary and secondary deviations facing the patient and visualize the angle of deflection and the limitation of movement in the affected areas, still he must revert his findings to the common position for comprehending them.

A strange contradiction is found in the use of Duane's tangent plane for plotting the double images. Although the observer is in front of the patient carrying the light and thrusting pins in the curtain, and although the graduation of the latter is turned toward the observer and the marks made in it show through, still he must consider the right and left side according to his own and not to the patient's sides.

In the detection, measurement, and correction of heterophorias the same thing happens. The tests must be conducted from before the patient, but the estimation of the defect and the thinking is done from behind.

For men who are accustomed to use the anatomic nomenclature the reading in the text-books of the chapters on paralysis of the ocular muscles, and the transfer and memorizing of symptoms in the proper position, require a long, painful, and strained mental process, which will be made unnecessary if one standard position both for objective and subjective examination be used (Fig. 1).

This does not mean that the observations and tests upon our own

eye should be entirely discarded, but only that they shall constitute the exception and not the rule as they are to-day. Of course, many ophthalmologists already trained in this school of double positions, and who have attained the habit of the mental processes required to make the inversion quickly and without effort, will find it entirely unnecessary or even inadvisable to depart from their habits, but for the beginner in ophthalmology to be taught in the orthodox way shall mean much time saved and much effort spared.

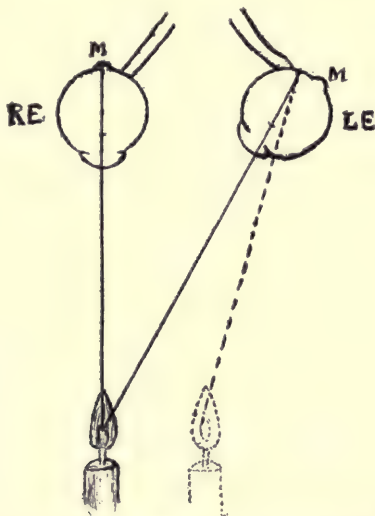


Fig. 1.—Homonymous diplopia. The deviation of the left eye inward makes the image of the candle fall at the inner side of the macula, M, and is projected outward, where a false image is seen at the left.

THE INVERSION OF THE VISUAL FIELD

The ordinary way of recording the visual fields is to consider the findings as seen from an observer at the back of the patient. This was probably made necessary in the beginning by the use of the black-board or campimeter, in which the limits were drawn up as if made by the patient's hand. In this position the visual field of the left eye faces the left side of the patient and has the normal notch corresponding to the nose downward and inward, opposite to the corresponding part of the right side (Fig. 2).

With the advent of the perimeter, however, the interpretation of the findings became more difficult, because the observer, standing in

front of the patient and watching his eye for a correct central fixation, needs to transfer his findings from the arc to a chart and use a mental operation to determine on which side the marking ought to be done. To facilitate the recording and make it a mechanical operation the self-registering perimeter was devised. In this instrument the findings are marked on the opposite side of the arc, upon a graduated scale, which has the same color of the figures on the arc.

The violation of the anatomic standard which this recording of the field entailed made imperative for the understanding of the subject proper orientation of the oculist to extend this same faulty position to

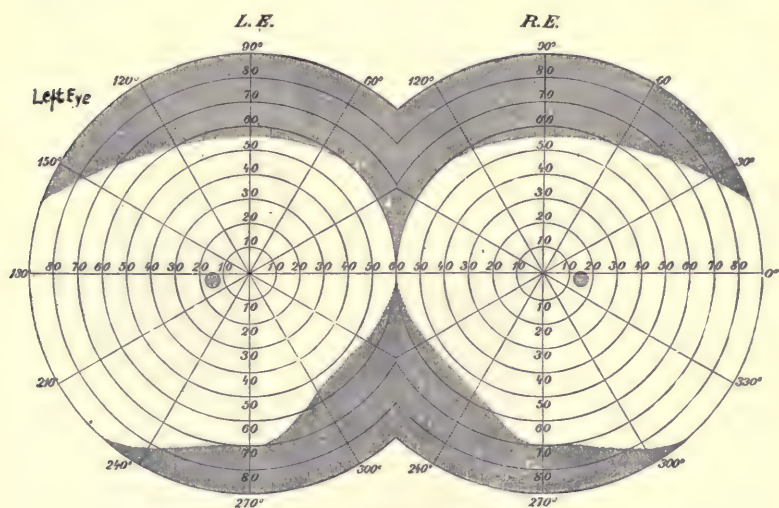


Fig. 2.—The fields of vision as recorded at present.

the anatomy, physiology, and neurology of the eye, setting two different standards for the description of the organs, lesions, and functions.

The result of this great departure is an added mental strain in order to transpose the mental images from the patient in front of the observer to the field, and determine, for instance, to which part of the retina one scotoma, a sector-like defect, etc., corresponds. In case of a thrombosis of the superior temporal artery of the right retina, where will the sector-like defect of the field correspond? Undoubtedly to the inferior nasal quadrant. The student visualizing the ophthalmoscopic lesion and wishing to represent to himself the defect on the

field needs to transfer the lesion first to his own eye and then find the normal inversion in the field and locate the defect.

The understanding of certain complicated lesions in the brain and the impairment of the functions they produce are more difficult to visualize with the present standard.

As we have pointed out before, in case of hemianopsia the correla-

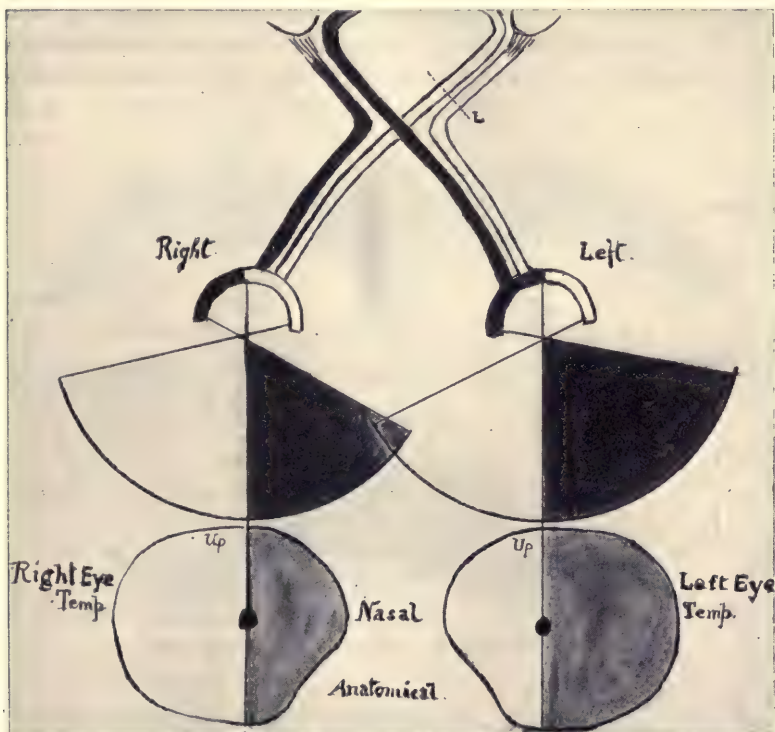


Fig. 3.—Schematic representation of the optic pathways, fields of vision on both sides as projected in the space in front of the observer, and records of the fields in the anatomic position.

tion of the blind sides of the retinae with the visual fields and the patient's right or left is very difficult.

If a lesion is present in the left optic tract, the text-book tells the student that the left halves of both retinae shall be wanting; the patient will see only the left halves of all objects. In the clinic the patient is facing us and explaining which side of the objects he sees;

by the perimetric examination we shall determine that the temporal field in the right eye of the patient and the nasal field of the left are wanting, still we must record the defect referring all these symptoms to our own eye and make the diagnosis by ascertaining which side of the objects our hemianopic eye would see, then we will call the hemianopsia right or left sided.

Would it not be easier to follow the anatomic rules and record the fields as projected into the space in front of the subject, the right field in front of his right eye and the left before his left? (Fig. 3.) We would look at them as we see the fundus, as we really found them when moving the test object upon the arc of the perimeter.

In this way the transfer to our own eye will be useless and no mental operations shall be necessary to visualize the field as located in space.

This new method has many advantages: In the first place, the understanding of the normal retinal projection with the field mapped out in space, in the opposite side of the retina, and the difference between the size of the temporal and nasal sides of the field, is made much easier. The extension farther forward of the sensitive portion of the retina on the nasal side, and the crossing of the limiting rays in the nodal point of the eye, can be demonstrated more clearly.

The localization of the blind spot and paracentral scotomas in relation with the visual axis is much easier to understand, as we are more accustomed to visualize the relations of the disc and the macula in the fundus with the ophthalmoscope.

The reference of the ophthalmoscopic and neurologic lesions to the sides and places in the fields are greatly simplified by *one* physiologic inversion only instead of the two now in use.

There are no technical difficulties in the change from the old to the new anatomic standard. The ordinary campimeter can be made of cloth instead of board, and with a graduation in the back which will allow, by thrusting pins, the reading of the extent of the field in front of the patient.

The Bjerrum screen, as now made, will be more easily used, no transfer of the readings in the back to the present unanatomic standard being necessary; this transfer and also recording from the campimeter being a real difficulty for the beginner.

With the perimeter the only changes will be first to print the same schema now in use not exactly inverted but as seen by translucency; the temporal side in front of the temporal side of the patient's orbit in each side, and the nasal side before the nasal side of the orbits (Fig. 4).

The positions of the test-carrier in the arc shall be marked on the same side of the recording scale, the temporal findings in the temporal side of the scale, and so on.

In order to avoid the confusion which in the beginning falls to the lot of any change, the charts will be left as they are now, only printing on the back another schema, with the graduation and limits as seen by translucency. A short direction printed in them will direct the operator to pencil the markings in the same side of the graduated scale; temporal side of arc on outer part of the scale, nasal on inner side.

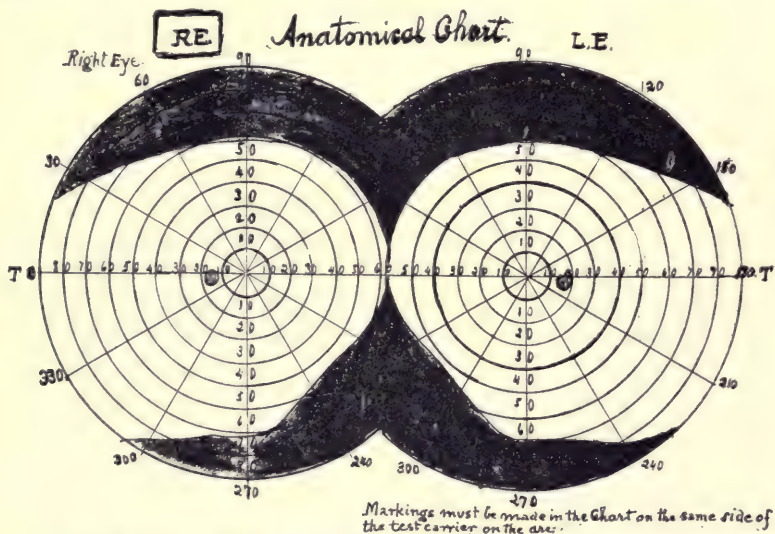


Fig. 4.—The fields of vision as recorded in the anatomic position.

The above reasons and the numerous advantages to be derived from a change in the present position and in the field of vision afford us the opportunity and honor of submitting to the International Congress of Ophthalmology of Washington, D. C., the following proposition:

The International Congress of Ophthalmology, held in Washington, U. S. A., recommends to the authors and editors of books and essays dealing with ophthalmology to adhere faithfully to the anatomic nomenclature, and always consider the subject standing or lying down in front of the observer, his right side facing the left hand of the latter.

This rule to be applied both to objective and subjective examinations of the eye and also to the visual field.

STANDARDIZATION OF PERIMETRIC TECHNIC

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The variable elements in field taking can be included in two groups: First, those which are correctable and due to a lack of care on the part of the operator, on the part of the patient, or to inadequacy of the necessary equipment; and, second, those which tend to yield variable results from day to day because of organic or functional changes in the visual pathway.

By perimetric study we endeavor to uncover the latter group for correlation with other symptoms. In order, however, to be of dependable value, the errors of the first group must be eliminated. To this end efforts have been made from time to time to develop a technic which can be made uniform, or approximately so, throughout the world, or wherever perimetry is practised. Notwithstanding these efforts the frequent mention in literature of the need of standards in our methods, and of urgent appeals by individuals for uniformity, the first group of variable factors continues to rob field studies of their real value, although these errors can, for the most part, be eliminated.

In order, therefore, to approach this subject from an international standpoint, and to secure the co-operation of ophthalmologists from the countries here represented, the following resolution is respectfully submitted:

Resolved, That a Committee be appointed by the President of this Congress of Ophthalmology now assembled, to consider and to recommend to this Congress, before its close, a standard in technic which may be applicable in any part of the civilized world.

First: With reference to the designation of the test objects or stimuli in degrees, minutes, or seconds.

Second: The designation of the pigments, or papers, to be employed as colored stimuli in definite and understandable language, by which they can be reproduced by the manufacturers of perimetric instruments.

Third: Uniformity in charts.

Fourth: A standard method of illumination.

Fifth: A method by which pre-exposure and surrounding field may be applied to perimetric studies.

Sixth: The use of instruments in field studies with special reference to the needs of the case.

Seventh: Other recommendations which, in the judgment of the Committee, may seem advisable at this time.

ADDRESSES DELIVERED BEFORE THE CONGRESS

SOME DESCRIPTIVE ERRORS IN THE ANATOMY OF THE ORBIT

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The points briefly described in the address were illustrated by lantern slides of figures from various text-books compared with photographs of actual dissections. It is not feasible to reproduce them here, but the figures are referred to in the text, and the photographs will be found in the writer's recent work on the anatomy of this region.

A comparison of the figures and descriptions of certain features in the eyelids, lacrimal apparatus, and orbital muscles, as given in the current anatomic text-books and copied thence into special ophthalmologic works, with what may easily be shown by dissection to be the actual formations, shows that some revision of the subject is called for. In addition, a general acceptance of one term instead of the vicarious application of several names to the same structure may well be urged.

The following points in particular seem worthy of notice; they are put forward in no hostile criticism of our text-books, but as the writer's experience in dissection of a considerable number of orbits.

(1) The ligaments by means of which the conjoint extremities of the tarsal plates of the eyelids are attached to the orbital margin are referred to in the texts by various names, and are not accurately described. They should properly be called **TARSAL LIGAMENTS**, medial and lateral, since the term "palpebral" in connection with such composite structures as the eyelids is somewhat indefinite; the expression "palpebral ligament," moreover, has been applied by some authors to the septum orbitale.

The **MEDIAL TARSAL LIGAMENT** (the ligamentum palpebrale mediale of the B.N.A. terminology, also known as the tendo oculi internus, orbicular tendon, internal palpebral or tarsal ligament) is always illustrated as a band with well-defined upper and lower margins, crossing over the middle of a bulging lacrimal sac on its way to gain attachment to the anterior lacrimal crest and frontal process of

the maxilla, as in Gray, Fig. 938; Fuchs, Fig. 285; Cunningham's Text-book, Fig. 699, and Practical Manual, vol. iii, Fig. 7; Piersol, Fig. 1199; Sappey, vol. iii, Fig. 665; Merkel, Fig. 38; Spalteholz Atlas, Fig. 929; Sobotta-McMurrich Atlas, Fig. 763; and Testut, vol. iii, Fig. 494, which is reproduced in many books.

Actually, the medial tarsal ligament forms a complete anterior covering to the whole of the upper half of the lacrimal sac. Superiorly it is thin and spreads out to blend with the periosteum of the bone, so that any definition of an upper margin appears to the writer quite artificial; this fact can easily be verified by removal of the skin over the medial commissure of the eyelids, and then dissecting away the fibers of the orbicularis oculi muscle which arise from the face of the ligament. Inferiorly, however, the ligament does present a conspicuously free and thick lower margin; this edge lies on a more anterior plane than the rest of the somewhat obliquely disposed ligament, and is denude of muscle fibers, facts which account for its prominence beneath the skin in the living, and its accentuation by lateral traction of the eyelids. Satisfactory definition of a posterior or reflected portion of this ligament, passing behind the lacrimal sac (as figured by Testut, vol. i, Fig. 697; Poirier, Fig. 685; Sobotta, Fig. 763), is difficult; it is very thin and appears of quite secondary importance, being merely the fascia clothing the front of the pars lacrimalis muscle. Sometimes it is apparently confused with the lacrimal fascia (vide infra).

The terms LATERAL TARSAL or PALPEBRAL LIGAMENT (*tendo oculi externus*, *ligamentum canthi externum*) and RAPHÉ PALPEBRALIS LATERALIS (B.N.A.) are variously and confusedly applied, both in text and illustration, to two entirely different and separate structures, of which only the one or the other is recognized, whereas both are actually demonstrable. Extending between the lateral canthus of the eyelids and the orbital margin there is an actual RAPHÉ, formed by the interlacing fibers of the palpebral portion of the orbicularis oculi muscle and strengthened upon its deeper aspect by the fascial septum orbitale, but it passes superficial to the orbital margin and is only adherent to the bone by connective tissue. On cutting through this raphé, the tarsal plates, though more movable, will be found still attached to bone by a deeper lying, much stronger and more definite band of connective-tissue fibers, which is the true lateral tarsal LIGAMENT. It is separated from the superficially lying raphé by a narrow but usually definite cleft, in which there may be present a few lobules

prolonged downward from the lacrimal gland; and its actual attachment is just within the orbital margin, onto the orbital tubercle of the zygomatic bone. Some books figure the muscular raphé correctly in one place, but misapply the name to what is evidently the ligament in another; they also show the ligament as passing, like the raphé, superficial to the orbital margin instead of within or posterior to it (compare Figs. 340 and 929 in Spalteholz). Both structures are illustrated in Piersol, Figs. 500 and 1199, but the text denies the existence of the ligament on p. 484. Cunningham misapplies the terms in the text-book, Fig. 699, and practical manual, vol. iii, Fig. 7; as does also Sobotta, Fig. 763. Fuchs alone describes both correctly (p. 650).

With further reference to the TARSAL PLATES, it may be noted as an example of the longevity of anatomic descriptive errors that the term "tarsal cartilage" still occasionally persists (Young's Handbook, fifth edition, p. 373; Howe, vol. i, pp. 15 and 16); though C. Krause pointed out as long ago as 1842 that no cartilage cells are present. In spite of the firm consistency of these plates, the most striking feature seen in microscopic sections is the relatively enormous size of the contained tarsal or meibomian glands; indeed, there appears more glandular formation present than supporting connective-tissue framework.

As further terminologic inexactitudes it may be noted that the "glandulæ mucosæ Krausei" of the B.N.A. are not mucous glands, but are accessory lacrimal glands of the conjunctiva. Also that Zeis, who apparently first described in 1835 the sebaceous glands of the eyelid margins known by his name, spelled the latter as above, and not, as generally given, Zeiss.

(2) The LACRIMAL FASCIA (fascia lacrimalis) is an extension of the periosteal (periorbital) lining of the medial wall of the orbit which passes from the posterior to the anterior lacrimal crest, bridging over completely the lacrimal fossa or groove with its contained lacrimal sac. Its presence must surely be well recognized by and be of interest to the practising ophthalmologist, yet the writer has found no figure and but rare mention of it in the books, and even then it is referred to by such misleading terms as the "deep" or even "palpebral" fascia (e. g., Schaeffer, p. 249); sometimes it appears to be confused with the deep or reflected lamella of the medial tarsal ligament (Toldt, Fig. 1393; Morris, p. 1106; Fuchs, Fig. 284). It is a strong definite layer of fibrous tissue, and forms the immediate lateral and complete covering of the lacrimal sac; it is pierced by the lacrimal canaliculi and blood-vessels. Occasionally the fascia is adherent to the sac, but it can

usually be incised separately in gaining entrance to the lumen of the latter; failure to realize this point in passing a probe commonly leads the student in the dissecting room to force the instrument downward between the incised fascial covering and the unopened sac, and naturally a difficulty is experienced in reaching the nasal cavity. The extent and attachments of the fascia and the manner in which it completely roofs over the fossa can best be demonstrated by dissecting out the entire nasolacrimal duct and lacrimal sac from the nasal side; that is, by cutting away the medial bony walls of these passages by appropriate working from the inside of the lateral wall of the nasal cavity in a sectioned head (as illustrated by Schaeffer, Fig. 175). After severing the lacrimal canaliculi and freeing the fundus of the sac, the entire tube formed by the sac and duct can be lifted out from the excavation in one piece; and removal of the whole contents of the orbit with detachment of the eyelids on the opposite or orbital side of the preparation will leave the lacrimal fascia as a complete entity, exposed on both sides.

(3) The *PARS LACRIMALIS MUSCLE* (the muscle of Horner, 1824, but chronologically more correctly designated as that of Duverney, who previously described it in 1749, and well named it the tensor tarsi; it is also known as the posterior lacrimal muscle). The books nearly all follow the account given by Horner, who described the muscle as passing from its origin on the lacrimal bone forward and outward to divide and terminate near the puncta lacrymalia of the eyelids (e. g., Morris, p. 391; Testut, vol. i, p. 732; Howe, vol. i, p. 51, calls it a "supernumerary" muscle, and gives an insertion into the conjunctiva). The customary method of displaying it is from behind, by dissecting both eyelids away from the front of the eyeball and turning them nasalward, as depicted in Gray, Fig. 520; Piersol, Fig. 500; Sobotta, Fig. 261, and in Horner's original figure; so viewed, the *pars lacrimalis* fibers of the *orbicularis oculi* certainly appear to end as described near the lacrimal puncta. If, however, the tarsal plates be now dissected away (as shown, for example, in Spalteholz, Fig. 340), it will be realized that whilst some of the muscle fibers are short and do end on the canaliculi and tarsal plates, yet the bulk of them are continued on across the front face of the plates as far laterally as the lateral raphé. In other words, the *pars lacrimalis* or Horner's muscle appears to the writer to denote merely the conjoint ends of two continuous long muscle bands (the pretarsal part of the *pars palpebralis* of the *orbicularis oculi*) which sweep across the eyelids from one side

to the other, lying in front of and closely applied to the tarsal plates. They can thus serve far better to keep the latter curved and approximated in their whole length to the eyeball under all its movements, than if they were merely inserted into the medial ends of the plates. The continuity of the fibers and their passage at the medial end behind the lacrimal sac would explain why removal of the medial tarsal ligament in ablation of the lacrimal sac does not necessarily entail ectropion of the lower lid, as suggested by the writer in a paper in "The Ophthalmoscope," April, 1913. It may be noted that the ligament passes across the front of the upper half of the lacrimal sac, whilst the pars lacrimalis muscle passes behind its upper half, so that the two form a complete hood over the fundus of the sac. The lower half of the sac is related to the orbital fat behind, and is merely covered in front by septum orbitale, orbicularis oculi muscle, and skin.

(4) The shape of the normal LACRIMAL SAC appears, according to the writer's findings, wrongly represented in every instance. In all the figures enumerated in reference to the medial tarsal ligament in the first part of this article, the sac is drawn as seen from the front in the form of the bulging or dilated upper end of the nasolacrimal duct. Possibly this is due to the exigences of pictorial representation, though it is also so described in some texts (e. g., Quain, Schaeffer, Sobotta). Actually the sac conforms in shape to the bony fossa or groove in which it lies, and tapers off above, though its anteroposterior diameter in its middle may exceed that of the duct below. The shape of the whole tube, lacrimal sac and nasolacrimal duct together, may be likened to the end of a quill-pen seen sideways, a configuration that the writer has always found, especially in formalin hardened dissections prepared as described above with reference to the lacrimal fascia, and as is shown by Aubaret's series of isolated lacrimal sacs, and in the casts made by Zabel. Toldt's Fig. 1393 shows well the narrow transverse lumen of the sac in section, and will enable one to realize that any action which the fibers of the orbicularis oculi muscle may have upon it in the act of winking must be transmitted through the agency of the covering lacrimal fascia, and will tend to dilate the sac, which is possible from its collapsed condition (and, as indeed Scimemi proved experimentally) rather than to compress it, which does not appear possible either from its position or its condition.

(5) The LEVATOR PALPEBRÆ SUPERIORIS MUSCLE is oftentimes described as having a threefold insertion into the upper eyelid, namely to the skin, the tarsal plate and the conjunctiva, but it is

misleading to depict all three insertions as being of equal importance (e. g., Cunningham's Practical Manual, Fig. 8 and p. 26). The primary and essential attachment is to the skin, through the agency of the splayed out anterior margin of the expanded aponeurosis in which the muscle belly ends. The tarsal attachment is effected by the agency of the involuntary superior tarsal or palpebral muscle of Müller, which, though in close contact with the under surface of the aponeurosis and springing like it from the fore end of the belly of the muscle, does not "largely form part of it" (Piersol, p. 502); it is an extremely interesting little muscle, from a clinical standpoint as well as from a morphologic one, since its tone may be strikingly responsive to the state of the sympathetic nervous system by means of which it is supplied; and it may indicate by a slight drooping of the eyelid a condition of malaise (as is well seen in children) or of mental fatigue beyond the power of the will to counteract. The third so-called insertion of the levator is merely the attachment of its fascial sheath, fused with that of the subjacent superior rectus muscle and void of striated muscle fibers, to the superior conjunctival fornix. Its action is to pull this conjunctival fold upwards in harmony with movement of the cornea in the corresponding direction, and it might more fittingly be described as an additional "insertion" of the superior rectus, comparable to the similar fascial slips derived from the sheaths of the other recti muscles which pass to the conjunctival fornix below, lateral, and medial to the eyeball. Most of the illustrations give a complicated and indefinite view of these insertions; Spalteholz, Fig. 923, is the clearest.

Lastly, in descriptions of the levator, the osseous attachments of the lateral extremities or horns of the aponeurosis surely deserve more notice than they generally receive; the especial strength of the lateral horn, its enfolding by the lacrimal gland, and its strong fixation to the orbital tubercle of the zygomatic bone might well be mentioned.

(6) The LACRIMAL GLAND is described as being enclosed in a definite capsule (e. g., Poirier, vol. iii, p. 1122; Morris, pp. 1106 and 1113), but the writer has never been able to satisfy himself as to the existence of such a structure. The lobules of the gland are bound together by a connective tissue stroma which is certainly continuous behind with the interlobular fascia of the orbital fat; it is slightly more abundant upon the surface of the gland, where a delicate tissue can be picked off piecemeal, but such formation can hardly be considered as a "capsule" in the usual acceptance of the term. Nor can "suspensory ligaments"

be satisfactorily demonstrated. The gland is kept in place by the eyeball below and medially, by the orbital fat behind, but chiefly by the fact, usually not mentioned, that it is cut deeply into and so partly subdivided into its two lobes (orbital and palpebral portions) by the lateral horn of the aponeurosis of the levator palpebræ superioris muscle; it is, as it were, folded round the lateral part of the aponeurosis, and may possibly participate to some extent in its movements.

(7) The LATERAL RECTUS MUSCLE of the eyeball is commonly described as arising by "two heads" from the annulus communis of Zinn at the apex of the orbit, and certain nerves are described as entering the orbit between them. The statement conveys an exaggerated idea of the muscle origin, reflected in the figures where two definite and separate heads are depicted (e. g., Gray, Fig. 933; Cunningham's Practical Manual, vol. iii, Fig. 96). The muscle certainly arises from that part of the annulus which crosses the superior orbital fissure, but, as Dwight points out (p. 96), there is no break in the continuity of the annular origin of the muscle fibers, such as the expression "two heads" denotes. The term is probably a subversion of Merkel's description of one head arising from the annulus and another from the spina recti lateralis of the sphenoidal apex of the lateral orbital wall; he shows these two origins clearly (Fig. 28), as does Spalteholz (Fig. 916), but they do not span the superior orbital fissure, no structures enter the orbit through them, and they are not the same "heads" as described in later works. Merkel described the nerves which here enter the orbit as traversing an "oculomotor foramen," which is a definite fibrous ring formed between the annular origin of the lateral rectus muscle and the optic nerve. The position of the nerves (the oculomotor in two divisions, the nasociliary and the abducent) relative to one another at this point receives unwarrantable stress in the text-books (e. g., Buchanan, p. 1149), since they are all crowded together and compactly fill a space not more than three millimeters in diameter. It would appear better to describe these nerves as entering the orbit through either the oculomotor foramen, or between the lateral rectus muscle and the optic nerve (as does Sobotta), or within the cone of recti muscles which arise from the annulus, since the practical point is that the trochlear, frontal, and lacrimal nerves enter outside the cone of muscles.

(8) The FASCIA BULBI (Tenon's capsule) will lose much of its descriptive complexity if simply regarded as the primary socket of the eyeball, fused with the fascial sheaths of the muscles where they pierce

it. The elaborate description of Motais (followed by Maddox and Howe, and formerly included, but now replaced, in Poirier's *Anatomy*) forces one to agree with Dwight that the "complications of this membrane are limited only by the perverted ingenuity of those who describe it." The thickening of the capsule below to form Lockwood's ligament, and the peripheral expansions of the muscle sheaths to form "check ligaments" are easily demonstrable, but the formation of "pulley-bars" at the points of junction of the muscle sheaths with the capsule to prevent compression of the eyeball in contraction of the recti (Fisher, Fig. 14; Morris, p. 1107; Cunningham's *Practical Manual*, vol. iii, p. 260) is surely uncalled for if one considers the absence of angle or even curve in the run of a muscle at such point, together with the extreme mobility of the eyeball. The writer cannot find evidence that the capsule is continuous behind with any prolongation around the optic nerve, or that the interfascial space of Tenon, which separates the capsule from the eyeball, is continuous with the so-called supravaginal space along the nerve, as depicted in Piersol, Fig. 800, and elsewhere described. The eyeball cannot move freely within the capsule, as it is sometimes stated to do (Morris, p. 1107; Cunningham's *Practical Manual*, vol. iii, p. 259; Buchanan, p. 1151); there can be only limited movement between them, and both work together on the bed of orbital fat, as is agreed by Dwight, Fisher, and, indeed, by Motais. The point might be illustrated by comparing the capsule to a closely fitting woolen shirt, tightly buttoned round the neck (the sclero-corneal junction) and at the ends of the sleeves (the muscle sheaths), and so allowing no really free movement of the body ensheathed by it.

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ON THE CLINICAL ANATOMY OF THE EFFERENT LACRIMAL PASSAGEWAYS

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I take it as axiomatic that of first importance is a knowledge of the basic plan of the anatomy of the human body and that, in the diagnosis, treatment and prognosis of diseased states, the regions and organs of the body must needs be approached from the viewpoint of an average anatomy. It is fortunate that there is such a constancy in what may be considered basic, and the wonder is not that there are departures in anatomic conformations but that nature so regularly reproduces that which is fundamental. Despite the general truth of the foregoing an ideal or unvarying anatomy of many parts is uncommon. There are variations in essential details and the adherence to an arbitrary and so-called fixed normal is fraught with considerable danger, since with variations come altered size, shape and topographic relations; moreover, interiors and exteriors of parts are profoundly influenced by developmental potentialities. Anatomic variations of an organ and apparatus and of their relational field must have an important bearing in diagnosis, pathology, clinical medicine, and

surgery, and the attention given to this fact may be the narrow margin between success and failure. It is this thesis that I venture to support and develop in my address on the efferent lacrimal passageways this evening rather than enter into a detailed account of the conventional anatomy of the parts concerned.

GENESIS AND DEVELOPMENT

It would be futile to attempt a discussion of this problem without a brief presentation of the embryology of the efferent lacrimal apparatus. The genetic and developmental anatomy not only point the way to a clearer understanding of the ground plan of the anatomy, but of the anatomic types and anomalies as well.

In the early embryo, extending from the medial angle of the eye to the olfactory pit, is the naso-optic (nasolacrimal) groove or fissure, bounded above by the lateral nasal process and below by the maxillary process. These embryologic, mesenchymal and epithelial covered processes unite by coalescing from the depth toward the periphery, thus obliterating or outfolding the intervening groove or fissure. In 12 mm. human embryos, aged approximately five weeks, a ridge-like thickening of the deep layer of the epithelial lining of the now rudimentary naso-optic groove takes place. This epithelial ridge grows and pushes into the underlying mesenchyme and becomes entirely separated from its surface connections and wholly surrounded by mesenchymal tissue (embryos aged from thirty-six to forty days). The detached solid strand or cord of epithelial cells is the rudiment or anlage of the nasolacrimal passageways, and while it corresponds to the line of the previous naso-optic groove, it must be understood that the system of efferent lacrimal ducts does not represent a cut-off portion of the obliterated groove brought about by the coalescence of the lateral nasal and maxillary processes.

The rudiment of the nasolacrimal passageways is a solid cord of epithelial cells, detached from its genetic area and for a brief period entirely surrounded by mesenchyme and without a lumen. From the parent cord of cells sprout the upper and lower lacrimal ducts, the upper part of the lacrimal sac and the nasal end of the nasolacrimal duct, thereby establishing secondary connections with the epithelium of the free border of the eyelids and the nasal fossa. The lumen of the several segments of the nasolacrimal passageways is irregularly and variously formed. This is accomplished by an apparent necrobiosis and resorption of some central cells and a rearrangement of

others. Canalization begins early at the ocular end of the stem rudiment, progresses rapidly toward the nasal end and is well advanced in embryos aged one hundred days. At times it would appear that the upper (eye) and lower (nasal) ends of the stem rudiment were the first and the mid-section the last to gain a lumen. The horizontal

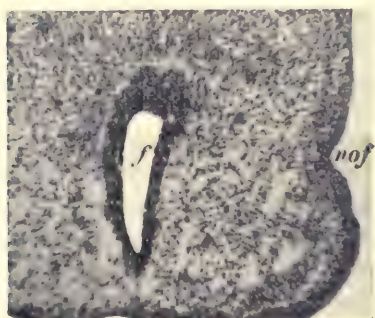


Fig. 1

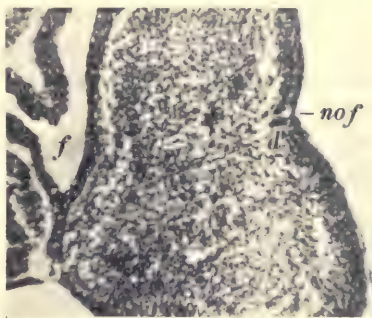


Fig. 2



Fig. 3

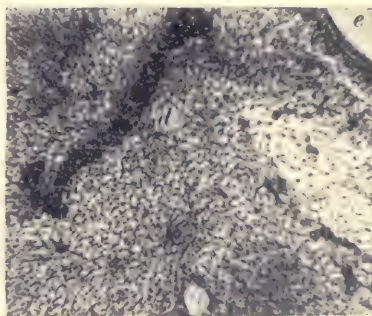


Fig. 4

Figs. 1, 2, 3, 4.—Photomicrographs of frontal sections of the heads of human embryos showing several stages in the development of the nasolacrimal passageways: Fig. 1, aged thirty-three days; Fig. 2, aged thirty-five days; Fig. 3, aged thirty-six days; Fig. 4, aged forty-three days. $\times 7$. (Schaeffer: *The Nose and Olfactory Organ*.)

nof, Remains of naso-optic furrow; *f*, nasal fossa; *e*, eye; *d*, different stages of the rudiment or anlage of the nasolacrimal passageways.

portions of the lacrimal ducts become canalized before the vertical portions. In embryos aged one hundred and twenty days the vertical segments not infrequently are solid cords of epithelial cells, while the horizontal segments already possess a lumen. The canalization of the points of union between the free border of the eyelids and the

lacrimal ducts is often deferred for a later period. The point of coalescence between the nasal end of the sprouting parent cord and the inferior nasal meatus is the last to become patent—the attenuated lacrimonasal membrane “rupturing” approximately at birth or later.

As stated elsewhere, the rudiment of the nasolacrimal passageways grows into the subjacent mesenchyme and becomes encompassed by it. The lacrimal ducts and the ocular and nasal ends of the nasolacrimal duct are secondary outgrowths and soon establish their definitive connections. Subsequently (third or fourth month) a stumpy cartilaginous process (*processus paranasalis*) arises from the lateral wall of the cartilaginous nasal capsule and surrounds a part of the developing nasolacrimal duct segment. The surrounding mesenchyme ossifies in the formation of the maxilla, the lacrimal bone and the inferior nasal conchal bone; the related cartilage usually disappears and the greater portion of the nasolacrimal duct ultimately becomes encased in bone, *the canalis nasolacrimalis*. The lacrimal sac comes to rest in an oblong, rounded osseous depression, the *fossa sacci lacrimalis*, while the lacrimal ducts occupy the soft tissues of the eyelids and a lower non-osseous portion of the nasolacrimal duct not infrequently courses in the mucosa of the lateral wall of the inferior nasal meatus.¹

ATYPICAL DEVELOPMENT

The foregoing recounts the origin and early development of what may be termed an ideal or so-called typical efferent lacrimal duct system. However, there is early evidence of variations from the ideal. From the genetic point of view the variations in the anatomy of the nasolacrimal passageways appear to fall into three important and more or less clearly defined groups: First, those that have their basis in rudiment potentials; second, those due to early arrests in the development of parts which should proceed from the stem rudiment, and third, those due to division of previously continuous ducts into discontinuous segments. The complete dissociation of the efferent lacrimal ducts, owing to marked monster formations, might be considered an unimportant fourth group, and deserving of no further mention in this connection.

¹For a more detailed account of the embryology see J. Parsons Schaeffer, “The Genesis and Development of the Nasolacrimal Passageways in Man,” *Amer. Jour. Anat.*, vol. xiii, 1912.

RUDIMENT POTENTIALS

Rudiment potentials are encountered which explain such variations and abnormalities as supernumerary lacrimal ducts for one or both eyelids; duplication, triplication, et cetera, of the lacrimal punctum; the slit and furrow lacrimal punctum; mucosal ledges and so-called valves within the nasolacrimal passageways; irregularities of contour; diverticula; variations in the anatomy of the lacrimal aperture in the inferior nasal meatus; osseous and membranous segments of the nasolacrimal duct, etc.

As a rule, but two sprouts proceed from the upper end of the parent or stem rudiment, one becoming the upper lacrimal duct and the other the lower lacrimal duct or canaliculus. However, occasionally one encounters multiple buds or sprouts, some of which develop sufficiently to establish definitive connections with the free border of the eyelids and become supernumerary lacrimal ducts. Others fail to establish connection with the eyelids and end blindly. The majority of these additional buds are resorbed or become small, blindly ending diverticula. Complete supernumerary lacrimal ducts also lead to multiple lacrimal puncta. However, at times in the normal number of lacrimal ducts, multiple puncta are encountered for one or both lids. These are readily accounted for by the fact that the lacrimal duct in coalescing with the free border of the eyelid may do so over a relatively extensive area. The canalization of this wide area of contact—the *lacrimo-conjunctival membrane*—may be slit-like, oval or fenestrated, the latter giving rise to multiple puncta. Indeed, the greater portion of the lacrimal duct or canaliculus may be represented by a furrow along the free border of the eyelid.

The canalization of the solid nasolacrimal rudiment occurs very irregularly and there is all evidence at an early time of mucosal ledges and valves and shallow diverticula. Secondary buds from the stem rudiment account for the larger diverticula, and the belief that these are acquired in adult life and the result of disease is not justified.

The variation in the topography and type of the nasolacrimal ostium or aperture in the inferior nasal meatus is cleared up by the embryology of the lacrimonasal membrane, formed by the fusion of the epithelium of the nasolacrimal duct with the mucous membrane of the nasal fossa. When the developing nasolacrimal duct comes in contact with the nasal mucosa at the roof and highest point of the inferior nasal meatus, the resulting ostium nasolacrimale is single,

large, unguarded by a mucosal valve, and, since it becomes surrounded by an osseous ring, stands permanently open. On the other hand, when the developing nasolacrimal duct establishes contact and fusion with the lateral wall of the inferior nasal meatus, the area may be a considerable one. In the latter, the ostium nasolacrimale generally is slit-like and has a mucosal flap or valve. This valve may or may not be efficient physiologically in preventing regurgitation. The aperture may be single or multiple and located variously on the lateral wall of the inferior nasal meatus, from the attached border of the inferior nasal concha to the floor of the nose. Varieties of these two basic types of nasolacrimal ostia or apertures are very frequently encountered, but a discussion of them is not vital in this connection.

The posterior lacrimal crista is very prominent in the newborn, rendering the lacrimal fossa relatively deep. The hamular process of the lacrimal bone at times undergoes conspicuous development, resulting in division of the lacrimal sac.

DEVELOPMENTAL ARRESTS

Patients are encountered with one or both lacrimal ducts wanting in whole or in part; or if complete, the point of coalescence with the eyelids may be imperforate, or atresias over greater segments may be encountered. The connection with the inferior nasal meatus may be defective or absolutely imperforate. Atresias of portions of the nasolacrimal passageways are not uncommon in the newborn.

The stem rudiment may not become detached wholly from its original surface connections, thereby leading to lacrimal fistulæ or false adult connections. This is especially prone to occur in the region of the lacrimal sac. The nasal end of the stem rudiment may fail to sprout or may establish connections with the middle nasal meatus by means of a side bud. Connections may be made with the lip, and the lower orifice of the nasolacrimal duct established there.

The cause for the failure of normal development is not clear. So far as the lacrimal ducts are concerned, there appears to be a critical period for them shortly after the detachment of the solid epithelial rudiment of the nasolacrimal passageways from its surface connection. If the lacrimal ducts which normally grow as secondary sprouts from the stem rudiment fail to show beginning growth shortly after this stage, there appears to be a great likelihood that they will not develop subsequently. Since the rudiments are solid epithelial cords, want of canalization will lead to atresias of various degrees and levels later.

These are most common at the distal or nasal end of the nasolacrimal duct in the newborn and child. It would appear that many of the latter atresias become perforate during the first weeks or months of extrauterine life. Atresias of the lacrimal ducts or canaliculi also are encountered. There also is some evidence of obliteration of a previously established lumen by a secondary proliferation of the epithelial lining of the ducts, especially the nasolacrimal duct.

OTHER ANOMALOUS CONDITIONS

There are a few anomalous conditions in the anatomy of the nasolacrimal passageways that appear to have no basis in the rudiment potentials nor in arrested development. For example, divided lacrimal ducts are encountered, the lacrimal sac may be detached from the main nasolacrimal duct, the nasolacrimal duct proper may be found as two discontinuous segments, etc. It appears certain that the division of a lacrimal duct or of the nasolacrimal duct proper occurs secondarily; that is, after the definitive connections are fully formed. These conditions often occur in monsters or in children with secondary facial fissures. Ask and van der Hoeve¹ suggest amniotic bands and ingeniously use secondary facial furrows and the development of the lacrimal caruncle in attempting to establish their thesis. Ask's previous conclusions on the development of the lacrimal caruncle are generally accepted as correct.² It is my belief, for example, that the lacrimal ducts wholly grow from the ocular end of the stem rudiment of the nasolacrimal passageways and not, as some argue, one part from the free border of the eyelid and the other from the stem rudiment. Therefore, when one encounters a lacrimal duct with normal definitive connections, divided into two discontinuous segments, there is no other explanation but that the duct suffered division secondarily. The same applies elsewhere. Amniotic bands are suggestive in this connection and are deserving of further study. The discussion, however, cannot be extended here.

¹ Fritz Ask und J. van der Hoeve: Beiträge zur Kenntnis der Entwicklung der Tränenröhrchen unter normalen und abnormen Verhältnissen, letzteres an Fällen von offener schräger Gesichtsspalte, von Graefe's Archiv für Ophthalmologie, cv, 1921.

² Ask, Fritz: Über die Entwicklung der Caruncula lacrimalis beim Menschen, nebst Bemerkungen über die Entwicklung der Tränenröhrchen und der Meibom'schen Drüsen, *Anatom. Anz.*, xxx, 1907.

EARLY POSTUTERINE DEVELOPMENT

The nasolacrimal duct at birth has very irregular walls. The ragged mucosal surface gradually changes to a more even contour. However, some of the recesses remain and others become extended into goodly sized diverticula. Some of the mucosal fringes undergo definite organization and form permanent mucosal ledges and valves within the ducts. Not infrequently the nasal end of the nasolacrimal duct is imperforate at birth. Many of these go unrecognized, becoming perforate in the early weeks of extrauterine life and showing



Fig. 5.—*a*, Photograph of a section through the nasal end of the nasolacrimal duct, showing a lacrimonasal membrane of considerable thickness and imperforate. Fetus aged seven months. *b*, Note lacrimonasal membrane thinned out but imperforate. Newborn child. *c*, Section of the entire nasolacrimal duct, showing great irregularity of lumen. The communication with the inferior nasal meatus is established. Child aged one month.

that developmental processes are carried on after birth. However, some remain imperforate for a longer period and give definite symptoms. They may become perforate spontaneously, even after the first year. While an expectant treatment should be adopted, operative procedures may be required.

The nasolacrimal duct at birth averages less than 2 mm. in diameter, at the third year 3 mm., with extremes of 1.5 mm. to 6 mm., the latter being rare. The osseous nasolacrimal canal is somewhat larger. The subepithelial tissue stratum harbors a plexus of veins and lymphoid tissue.

The outlet into the inferior nasal meatus is extremely variable at an early time and is subject to the same influences that obtain in the adult. Since these will be discussed at length subsequently, they need not detain us here.

The plane of direction, in the infant, of the lacrimal fossa and the nasolacrimal canal, with the contained lacrimal sac and the nasolacrimal duct, is more direct and more nearly vertical. As age advances the plane of projection of the canal and duct must conform to the type of face, size of the inferior meatus, width of the pyriform aperture and the width of the bridge of the nose, which take definition and become more and more positive as puberty is neared.

MACROSCOPIC ANATOMY

The Lacrimal Fossa and the Nasolacrimal Canal.—The osseous fossa which lodges the membranous lacrimal sac (fossa sacci lacrimalis) is an oblong, rounded depression located on the ventro-medial aspect of the orbit,—the frontal process of the maxilla and the lacrimal bone participating more or less equally in its formation. The fossa is limited dorsally by the posterior lacrimal crista and ventrally by the anterior lacrimal crista, the former a prominent crest on the lacrimal bone (especially conspicuous in the newborn) and the latter a crest on the frontal process of the maxilla. Above, the lacrimal fossa becomes shallower and shallower, ultimately losing its identity at the fronto-lacrimo-maxillary suture line, while below it is directly confluent with the osseous nasolacrimal canal. The latter terminates immediately below the attached border of the inferior nasal concha at the cupola of the inferior nasal meatus. The osseous canal is formed by the maxilla, the lacrimal bone and the inferior nasal concha, the maxilla by its frontal process and body contributing the greater portion. Variations occur.

The length of the osseous nasolacrimal canal does not necessarily conform to the enclosed membranous nasolacrimal duct, which is frequently longer (see elsewhere). The osseous canal proper varies in length from 10 to 20 mm., and the lacrimal fossa from 10 to 14 mm., making the total length of the osseous channels from 20 to 34 mm. The diameter of the osseous canal is seldom uniform throughout, measuring on an average 5 mm. at its narrowest part and 8 mm. at its widest. Extremely narrow (2 mm.) segments are occasionally encountered. At times the widest point is above and the narrow point at its inferior outlet. Again, the reverse may be true, or the nar-

row part be in the mid-section. These congenitally constricted segments simulate a stricture in passing the lacrimal probe.

The plane of direction of the osseous nasolacrimal canal obviously conforms to the type of the facial skeleton. The breadth of the bridge of the nose, the width of the inferior nasal meatus, and the degree of expansion of the pyriform aperture also influence the course or direction of the nasolacrimal canal. Generally speaking, one may say that the osseous nasolacrimal canal is projected caudalward, lateralward and dorsalward. Its surface mark is an oblique line erected from the lacrimal fossa to a variable point on the medial aspect of the alveolar process of the maxilla, corresponding to the interval between the second premolar and the first molar, the first and second molars or even as far dorsal as the second and third molar teeth. The almost vertical plane frequently shown in text-books is erroneous.

The Lacrimal Ducts.—The lacrimal ducts or canaliculi begin normally by minute openings, the *lacrimal puncta*, which either surmount or are placed on the sides of the conical lacrimal papillæ, located on the free border of the eyelids, the upper about 6 mm. and the lower about 8 mm. from the medial palpebral commissure (internal canthus). The puncta normally are directed against the eyeball, and should there be a malposition (congenital or acquired) of the puncta there is usually an overflow of tears. The same results if the lumen is obstructed. The lacrimal ducts consist of vertical and horizontal portions, with dilatations or ampullæ at the knees. They average from 8 to 10 mm. in their total length; 0.1 to 0.2 mm. in diameter at the puncta, 1 mm. at the knees and 0.5 to 0.8 mm. in the horizontal portion. The inferior lacrimal duct almost invariably is longer than the superior duct. Irregularities and sacculations of the lumen are not infrequent.

The horizontal portions of the lacrimal ducts communicate with the lacrimal sac (a) by the ducts uniting into a short, narrow common duct, (b) by each duct emptying separately into a diverticulum of the lacrimal sac and (c) by the ducts emptying separately directly into the lacrimal sac. The first is the most frequent.

The Lacrimal Sac and the Nasolacrimal Duct.—The membranous lacrimal sac occupies the lacrimal fossa and extends for some distance into the upper end of the nasolacrimal canal. It is bridged over by the palpebral fascia, which extends from the anterior to the posterior lacrimal crests; the medial palpebral ligament; fibers of the orbicularis palpebrarum and skin and subcutaneous tela. The

orifice (or orifices) of the lacrimal ducts usually is located from 2 to 5 mm. from the extreme top or fornix of the lacrimal sac.

The lacrimal sac at times merges imperceptibly with the nasolacrimal duct. Again, there may be a slight or marked constriction or isthmus, or the lacrimal sac and the nasolacrimal duct may not be in alignment, but joined side by side. It will be recalled that the osseous nasolacrimal canal terminates at the highest point of the inferior nasal meatus. The contained membranous nasolacrimal duct may or may not conform to this termination. At times the nasolacrimal duct is continued for a considerable distance within the mucous membrane of the lateral wall of the inferior nasal meatus, beyond the nasal end of the osseous nasolacrimal canal. This accounts for the discrepancy in length between the membranous nasolacrimal duct and the osseous nasolacrimal canal; moreover, leading to an osseous-supported segment of the duct and a purely membranous segment. The latter has an important clinical significance (see elsewhere).

Since the ostium of the nasolacrimal duct varies in its location, the nasolacrimal duct has a range in length from 10 to 28 mm. The diameter of the nasolacrimal duct is not uniform. At times the isthmus is much constricted, 3 mm., the remaining portion of the duct averaging approximately 5 mm. However, this is much reduced at times by encroachment of the osseous canal or by an unusual development of the subepithelial strata. The nasolacrimal duct may be wide above and narrow below, narrow above and wide below, or hour-glass in character, *i. e.*, constricted midway.

Diverticula and Valves.—A large number of nasolacrimal ducts have fairly regular and uniform walls. However, not infrequently there is a retention of developmental structures leading to the formation of valve-like folds and diverticula, with great irregularity of the lumen of the duct. Krause and Beraud long ago described a valve at the junction of the lacrimal sac and the lacrimal duct. Frequently this is merely an elevation of mucosa. Again, it may be sufficiently reduplicated to form a true valve. Bochdalek and the writer have found that the opening of the lacrimal ducts may be placed in the center of a mucous membrane diaphragm. Additional valves and mucosal ledges are not infrequently encountered in the mid-portion of the nasolacrimal duct.

Lateral evaginations of the mucosa result in minor depression and fossæ. However, at times these extensions are of considerable pro-

portions, leading to the formation of variously sized, blindly ending diverticula. These diverticula have a lining not unlike that of the nasolacrimal duct proper, with which they always communicate. The genesis of these diverticula seems established, and from the evidence at hand one must conclude that they are congenital rather than acquired.

The anomalies of the efferent passageways were briefly discussed in connection with the development, therefore, no further mention of them need be made here.

MICROSCOPIC ANATOMY

The *lacrimal ducts* have a lining of stratified squamous epithelium superimposed upon a sparse tunica propria rich in elastica. Fibers of the orbicularis oculi muscle lend strength and support, paralleling the horizontal segment of the ducts and encircling the vertical segment. The circular fibers are in essence a true sphincter and are so arranged that from the physiological point of view the lumen of the ducts is readily occluded. This may be brought about reflexly following excitation in the immediate neighborhood or at a remote and unsuspected point. There may, therefore, be an overflowing of tears from a physiologic obstruction of the very beginning of the efferent lacrimal passageways.

The *lacrimal sac* has a lining of columnar epithelial cells, usually arranged in a double layer. Here and there the surface cells are provided with cilia. Beneath the epithelium is a relatively thick fibro-elastic tunica propria with an abundance of lymphoid tissue, some of which is arranged in definite masses not unlike the aggregated nodules of the small intestine. Small tubular glands are always present; in some specimens in great abundance, in others but few are found. Not infrequently numerous elements of epithelium exhibit stages of conversion into mucus-containing goblet cells. These may be found over considerable stretches of epithelium. Here and there the epithelium becomes depressed and gland-like, goblet-cells lining the depressions. The tubular glands not infrequently contain goblet-cells.

Another important anatomic feature is the loose tissue which connects the tunica propria with the periosteum. This tissue everywhere is occupied by a rich plexus of veins which partakes of the nature of a modified erectile tissue. The tissue spaces are large. This stratum is subject to very rapid engorgement and depletion, and

under reflex and pathologic conditions may increase in thickness many fold. There is every reason that this stratum can be influenced reflexly owing to the excessive venous plexus. Complete physiologic obstruction of the lumen of the lacrimal sac may ensue from its engorgement.

The *nasolacrimal duct* also is lined with a columnar epithelium. Small tubular glands are encountered—most of the mucous type, others appear mucoserous in character. Between the epithelium and the

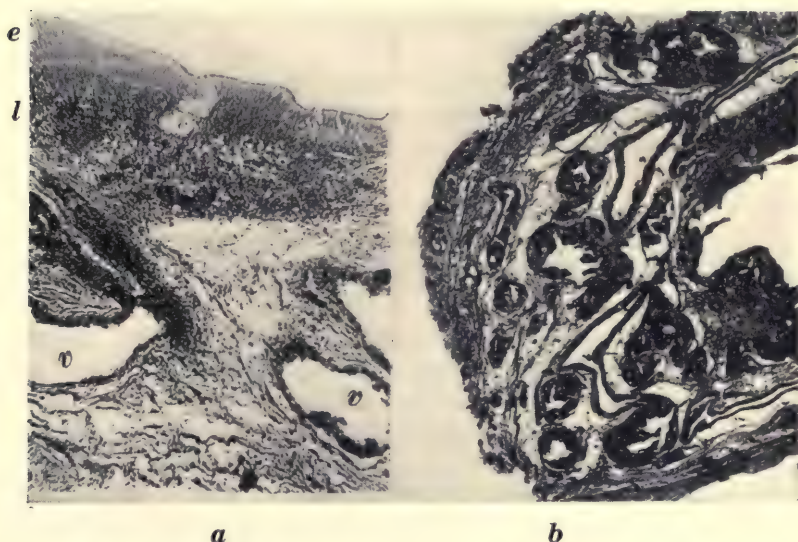


Fig. 6.—*a*, A section of the mucous membrane of the lacrimal sac, considerably magnified. Note, *e*, the epithelium with glands and goblet cells; *l*, lymphoid tissue, nodular-like in character; and, *v*, the large venous spaces. *b*, Same as *a*, at a lower magnification; showing a larger field and the great venous plexus in the tunica propria. The blood-spaces are all engorged.

periosteum is a stratum of very loose areolar tissue with an abundant venous plexus. The lower part of the duct is not unlike the erectile mucosa of the inferior nasal concha and the related meatus.

One cannot but be impressed with the abundant venous plexus of the lacrimal sac and the nasolacrimal duct. Its real significance has not been determined. At times and in places it is suggestive of an erectile tissue. One wonders whether this stratum of the lacrimal sac and the nasolacrimal duct is subject to the same influences as are the erectile tissues of the nose. The clinical potentialities here appear

great. Reflex neuroses may be a factor. However, in obscure cases careful search should be made for a disease focus which invokes a reflex turgescence and a possible physiologic occlusion of the nasolacrimal duct. It also is remotely possible that over-sexual activity may have a bearing as it does in nasal conditions. Of course, primary and secondary infection also markedly thickens the mucosa; especially is the venous plexus engorged and the lymphoid tissue multiplied. Another striking anatomic feature is the glands and the many mucus-containing goblet-cells. The anatomy is such that should the nasal outlet be interfered with a mucocele of the nasolacrimal duct and the lacrimal sac could readily follow. In view of the foregoing one wonders whether the lacrimal sac and the nasolacrimal duct are merely conveyors of fluid (tears) or whether an additional function should be ascribed.

THE EXTRAMURAL RELATIONS

The etiological connection between affections of the nasolacrimal passageways, the nasal fossæ and the paranasal sinuses is established beyond peradventure. This does not minimize the important anatomic and clinical connections between the efferent lacrimal passageways and the conjunctival culdesac. de Schweinitz says: "Although it might seem natural that conjunctivitis, and especially purulent conjunctivitis, should cause lacrimal disease, this is by no means frequently the case."¹ Kuhnt placed the nasal and paranasal origin of disease of the nasolacrimal passageways as high as 93.7 per cent. Others consider this too high. All, however, agree that the nasal cavity and its ancillary structures are fruitful fields from which disease spreads to the nasolacrimal passageways. Therefore, a brief consideration of the underlying anatomy is in order.

A. THE PARANASAL SINUSES

The Newborn and Child.—At birth the nasolacrimal duct lies approximately 2 mm. directly in front of and from 1½ to 2 mm. medial to the ventral end of the maxillary sinus. By the eighteenth month the distance intervening between the maxillary sinus and the nasolacrimal duct is reduced for a limited segment of the duct to an intimacy not unlike that in the adult. Later, when the infraorbital recess assumes larger proportions, the nasolacrimal duct and the maxillary sinus come into intimate relationship for a considerable distance. Of

¹ George E. de Schweinitz, *Diseases of the Eye*, 8th edition, page 569, 1916.

the ethmoidal cells, those developing from the frontal recess are nearest the lacrimal sac at birth. Topographic relationship is, however, not intimate at this time. Even at two years as much as 5 mm. intervenes between the lacrimal sac and the nearest ethmoidal cell, save in precocious development, when intimate relationships are established very early. In the sixth year the upper segment of the nasolacrimal duct and the maxillary sinus are intimately related, and the anterior ethmoidal cells (frontal and infundibular) have pneumatized the region formerly existing between the lacrimal sac and the frontal recess. Even the rudimentary frontal sinus may have close relationships at an early time. After the eighth year the anatomic relationships between the paranasal sinuses and the nasolacrimal passageways are in essence those of the adult.

The Adult.—The frontal and maxillary sinuses and the anterior group of ethmoidal cells need to be mentioned in this connection, and of these the latter are of special importance. Even the posterior ethmoidal cells when they pneumatize extensively forward into the middle nasal concha (conchal cells) may have a bearing in this connection.

Very commonly two or more ethmoidal cells which develop from the frontal recess and the ethmoidal infundibulum come into very intimate relationship with the dorsal and medial aspects of the lacrimal sac. Indeed, at times the entire lacrimal fossa and the upper part of the nasolacrimal canal are pneumatized by ethmoidal cells, variously related to the lacrimal sac and the upper part of the nasolacrimal duct. These ethmoidal cells for the most part arise from the ethmoidal infundibulum and the frontal recess and extend into the agger nasi, the uncinate process, the frontal process of the maxilla, and the lacrimal bone. While ethmoidal cells most frequently are located medial and dorsal to the lacrimal sac and the upper part of the nasolacrimal duct, they also are found cap-like over the cupola of the lacrimal sac and occasionally develop lateroventrally covering the lacrimal sac at the medial palpebral commissure. The expansion of the ethmoidal cells (anterior or posterior) into the middle nasal concha also is a factor. These readily become the seat of a mucocoele, pyocoele, etc.

The almost constant extension of ethmoidal cells from the frontal recess and the ethmoidal infundibulum on the nasal side of the lacrimal sac are of practical importance since dacryocystorhinostomy is practised in this location. Of course, when the nasolacrimal duct is

operated on in its lower third, the ethmoidal cells in question are avoided.

The osseous party walls between the lacrimal sac and the upper segment of the nasolacrimal duct and the ethmoidal cells are at best reduced to extremely thin and delicate lamellæ. Not infrequently the osseous partitions are partly or wholly defective whereby the mucoperiosteal layers of the related parts become contiguous.

The nasolacrimal duct courses in the osseous nasolacrimal canal,

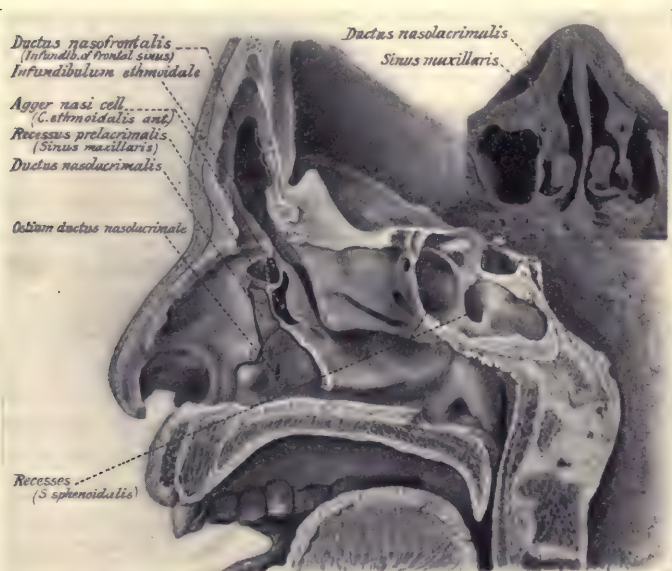


Fig. 7.—A dissection of the lateral nasal wall with especial reference to the nasolacrimal duct, the lacrimal sac (indicated by dotted outline, in white), the agger nasi cell, and the prelacrimal recess of the sinus maxillaris. (Schaeffer: The Nose and Olfactory Organ.)

located in the nasal wall of the maxillary sinus. Almost constantly this wall of the maxillary sinus is pushed into a bold, mound-like relief by the nasolacrimal canal and its contained duct. Another very important relationship of the maxillary sinus is the forward and cephalic expansion of the sinus in the formation of the prelacrimal or infraorbital recess. This recess is often of conspicuous size and hollows out the osseous boundary on the dorsal and nasal side of the lacrimal sac and more frequently of the nasolacrimal duct. Unless

one bears this expansion of the maxillary sinus in mind in surgical endonasal procedures, the sinus is readily opened into instead of the osseous nasolacrimal canal and the membranous duct. Where the recess is very extensive, opening into it may be unavoidable. As elsewhere, the osseous party walls, not infrequently, are extremely thin, even to the point of dehiscences.

The frontal sinus usually does not bear an intimate relationship to the lacrimal sac and the nasolacrimal duct. However, when the pneumatization extends into the nasal bone, the frontal process of the maxilla and the lacrimal bone, beyond the confines of the suture line separating these bones from the frontal, the frontal sinus bears a direct and at times a very intimate relationship to the lacrimal sac and the fossa which lodges it. That is, the frontal sinus extends into the walls of the lacrimal fossa. The osseous partition between the frontal sinus and the lacrimal sac is, as a rule, of considerable thickness. However, it may be reduced to a papery delicacy or be defective.

The paranasal sinuses, especially the anterior ethmoidal (frontal and infundibular), may develop to such an extent and in such direction as to encroach upon the lacrimal sac and the upper segment of the nasolacrimal duct and mechanically affect their lumen. If, however, the enlargement of the ethmoidal cells is wholly on the dorsal and medial aspects of the lacrimal sac the latter is crowded merely ventrally and laterally, since there is no bone to prevent bulging in this direction. The nasolacrimal duct may be profoundly influenced by the ethmoidal cells, the maxillary sinus and the conchal cells, since it is imprisoned in an osseous canal and is readily pinched by pressure or by cells pneumatizing its walls. Again, the lacrimal sac and the nasolacrimal duct may be encroached upon by the paranasal sinuses actually invading the walls of the lacrimal fossa and the nasolacrimal canal. The degree of this encroachment is variable and depends upon the number of cells differentiated, the direction of growth and the size attained. While the intimate topographic relations of the paranasal sinuses and the nasolacrimal passageways usually are of importance only in diseased states, the fact remains that occasionally in healthy cells and sinuses the encroachment on the lacrimal sac and the ocular end of the nasolacrimal duct is such as to mechanically obstruct to a greater or less degree the lumen of the passageways.

The incidence of nasal and paranasal disease is greater than that of the efferent lacrimal passageways. However, the direct or indirect transference of chronic disease from the paranasal sinuses to the tear

passageways is well established clinically. Apart from the intimate topographic relationships already referred to there are other contributing factors. The bony party walls frequently are extremely thin or web-like. Again, they may be fenestrated, or single, large congenital dehiscences may be encountered. The venous plexuses of the mucosa of the nasal fossa, the paranasal sinuses and the lacrimal sac and the nasolacrimal duct freely communicate. The lymphatic networks of the parts also connect up, and where the osseous party wall is fenestrated or defective, the abundant tissue spaces are in direct relation. The venous plexuses of the nasolacrimal passageways connect with the facial, infraorbital and ophthalmic veins and Zuckerkandl has described a lacrimonasal vein which perforates the lacrimal bone and connects with a goodly sized vessel emerging from the submucosal venous plexus of the anterior ethmoidal cells. These anatomic features explain the spread of infection from the nasal fossa and the paranasal sinuses to the nasolacrimal passageways and vice versa. Contiguous tissue spaces and planes of tissue may be involved in diseased states. The blood and lymph vascular systems also are factors. Thin, bony party walls may be perforated and pus foci established between the ethmoidal cells and the lacrimal sac, between the lacrimal sac and the lacrimal fossa, and between the lacrimal sac and the skin, the latter frequently resulting in the formation of fistulæ. Again, the party walls may break down entirely and an empyema of ethmoidal cells directly transferred to the lacrimal sac and duct. The opposite also occurs, whereby ethmoidal cell and maxillary sinus suppuration is secondary to diseased states of the efferent lacrimal passageways.

B. THE NASOLACRIMAL OSTIUM AND THE INFERIOR NASAL MEATUS

The nasolacrimal ostium or aperture in the inferior nasal meatus may, from the clinical viewpoint, be divided into five types: (1) those that fail of canalization, that is, the lacrimonasal membrane remains intact; (2) those of microscopic size, therefore wholly inadequate for the function intended; (3) those located at the highest point of the inferior nasal meatus and unguarded by a valve-like fold of mucous membrane and because of complete osseous walls are wide-mouthed and stand permanently open; (4) those located in the mucous membrane of the lateral wall of the inferior nasal meatus some distance below the attached border of the inferior nasal concha and guarded by a valve of mucous membrane which is both anatomically and

physiologically adequate and (5) those located as above in the mucous membrane of the lateral wall of the inferior nasal meatus and have a valve of mucous membrane, but which from the viewpoint of physiology is inadequate.

It is certain that many children are born with atresia of the nasal end of the nasolacrimal duct, owing to an intact or imperforate lacrimonasal membrane. It also is equally certain that a goodly number

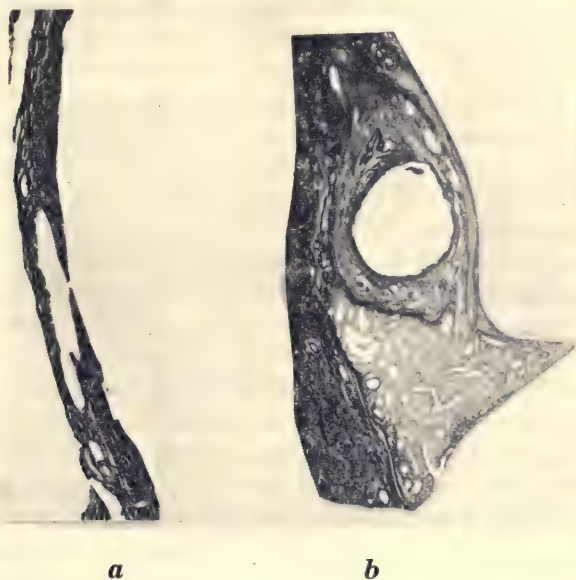


Fig. 8.—*a*, Type of nasolacrimal ostium located in the mucous membrane of the lateral wall of the inferior nasal meatus considerably below the highest point of the latter. Apertures in this position and of this nature are readily influenced by intranasal conditions. Note the valve-like folds guarding the slit-like orifice. Adult. *b*, Type of nasolacrimal ostium located at the highest point of the inferior nasal meatus, supported by an osseous ring and standing permanently open, unguarded by a mucosal valve. Adult.

of these persistent lacrimonasal membranes become attenuated and rupture spontaneously during the early weeks or months of infantile life. Indeed, at times it occurs much later. Of course, where the lacrimonasal membrane as such never formed; that is, where a goodly amount of connective tissue (of mesenchymal origin) persists between the epithelium of the nasolacrimal duct and the mucous membrane of the inferior nasal meatus, the barrier may be too thick for spon-

taneous rupture and surgical procedures often are necessary. This also is true in some instances of typical lacrimonasal membranes which are relatively thin, but for some reason or other do not "give way."¹ Again, epithelial debris may act as an occluding plug. Moreover, there is some ground for believing that previously established lacrimonasal communications occasionally become occluded secondarily by epithelial proliferation. Analogous processes occur elsewhere in the body; *e. g.*, the duodenum and the nares.

The nasolacrimal ostium when of microscopic size usually requires surgical attention. The small aperture may be the result of inadequate canalization or undue encroachment of the surrounding bone as it ossifies around the early nasolacrimal duct in the formation of the nasolacrimal canal. Not infrequently these small nasolacrimal ostia are located at the highest point of the inferior nasal meatus. Again, they may be located at a lower level in the mucosa of the lateral wall of the meatus.

There can be no doubt that the connection of the nasolacrimal duct with the inferior nasal meatus serves as a means whereby infection of the nasal fossa can extend into the nasolacrimal apparatus. Obviously the type of nasolacrimal ostium influences the incidence of occurrence of such extension and when once established the prognosis of speedy or delayed cure. When the nasolacrimal ostium is located high and supported by an osseous ring, causing it to stand widely and permanently open and unguarded by a valve of mucous membrane, it is possible for infectious air and fluids to pass into the nasolacrimal duct and from there into the lacrimal sac. Blowing of the nose in these cases readily drives particles of foreign material through the open and unguarded ostium into the duct and the sac beyond. Tobacco snuff has been known to ascend to the lacrimal puncta and cigar and cigarette smoke to make its escape from these apertures on the free border of the eyelids. The writer observed in a patient with a depressed fracture of the ventral or facial wall of the maxilla, hemorrhage from both lacrimal puncta. The nasolacrimal canal and the contained duct in the medial or nasal wall of the maxilla must have been injured. Fein reports a case of air escaping from the lacrimal puncta in which the nasolacrimal duct was injured in perforating the

¹ For an interesting and valuable clinical paper see William Zentmayer, Imperforation of the Lacrimonasal Duct in the Newborn and its Clinical Manifestations, Jour. Amer. Med. Assoc., vol. li, 1908.

maxillary sinus. It also has been observed in some cases of nose bleeding that blood escapes from the lacrimal puncta.

The same regurgitation of air and fluids from the inferior nasal meatus into the nasolacrimal duct may be argued when the ostium lacrimale is located in the mucosa of the lateral wall of the inferior meatus and has an anatomic valve but which is physiologically inadequate or insufficient. There is, however, a basic difference, since ostia in this position are not supported by osseous boundaries; moreover, the nasal end of the nasolacrimal duct courses for some distance in the nasal mucosa. Increased intranasal pressure would tend to

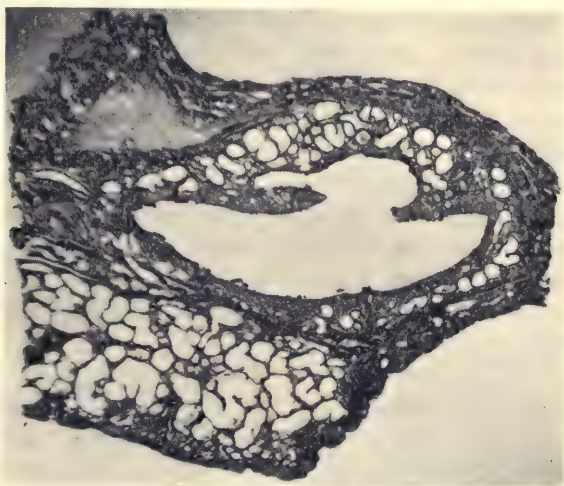


Fig. 9.—A section through the nasolacrimal ostium. Particularly note the cavernous or erectile character of the mucous membrane forming it and that it is not unlike the erectile tissue of the inferior concha adjacent to it.

collapse not only the nasolacrimal aperture, but the nasal end of the membranous duct as well, while the cases in which the nasolacrimal aperture is supported by bone and no membranous segment of the duct exists, would not be similarly influenced.

In such cases where the fold of mucous membrane at the outlet of the nasolacrimal duct is redundant, it not only may serve as an anatomic valve, but from the physiologic point of view be sufficient to prevent the regurgitation or passage of air and fluid from the nose into the nasolacrimal duct. It also may, to some degree, serve as a barrier to the extension of diseased processes from the nose to the nasolacrimal

apparatus. However, disease processes affecting the mucosa of the nose may extend into the nasolacrimal duct and sac despite the type of ostium, owing to the continuity of the mucous membranes, tissue spaces, etc.

It is but necessary to recall the location of the nasolacrimal ostium to comprehend why the aperture may be occluded by definite mechanical and pathologic states of the nose. Here again the type of ostium plays a conspicuous rôle. Hypertrophy of the inferior nasal concha may wholly or partially occlude the opening; the type supported by an osseous ring being less influenced than the one located in the mucous membrane of the lateral nasal wall and without bony support. Periostitis would affect the open-mouthed type of ostium and the duct in the neighborhood, but would not influence the actual ostium if located in the mucous membrane of the lateral wall some distance below the attached border of the inferior concha. Tumors, polypi, dental cysts, synechiæ, rhinoscleroma, etc., etc., readily influence the efficiency of the nasal outlet of the nasolacrimal duct.

It also is important to recall that the mucous membrane of the inferior nasal concha and the related lateral nasal wall and the ostium of the nasolacrimal duct frequently is of an erectile character. A mucosa of this sort is readily influenced reflexly and directly by diseased states.

DISCUSSION

The origin and development of the efferent lacrimal passageways point the way to a better understanding of the congenital variations and defects. A knowledge of the developmental history and potentialities is a distinct aid in deciding upon the course of the treatment in the several types of congenitally atypical ducts.

Fortunately the development of the efferent lacrimal or nasolacrimal passageways (lacrimal ducts, lacrimal sac, nasolacrimal duct) usually proceeds along methodical rather than fortuitous lines. Despite the general truth of the foregoing it does not necessarily mean a constant and unvarying anatomy in the adult; variations in essential details are not uncommon. The more important variations are subject to grouping into normal anatomic types and it is the thought of anatomic types and anomalies rather than the idea of an unvarying typical anatomy and anomalies that should be kept in mind when considering the applied or clinical anatomy of the efferent tear ducts.

There can be no doubt that the important anatomic types or departures from the so-called typical have a bearing in disease, prog-

nosis and treatment. Once infected and other factors being equal, it would appear established from an anatomic point of view that the lacrimal sac and the nasolacrimal duct free of mucosal ledges, valve-



Fig. 10.—*a*, A nasolacrimal duct (adult) with an unusually large, blindly ending diverticulum; *b*, a transection of an adult nasolacrimal duct in the region of a goodly sized diverticulum.

like formations and blindly ending diverticula; moreover, with a large open-mouthed and unobstructed connection with the inferior nasal meatus would give rise to less severe symptoms and yield more

readily and promptly to treatment; while the passageways with irregular walls, mucosal ledges and valves, diverticula of various degrees and an inadequate nasal communication would give rise to maximum symptoms, resist treatment, enter the stage of chronicity and, in all likelihood, require surgical interference. Granting the same type of infection and degree of involvement, the prognosis for a speedy and permanent cure in the two types of the efferent lacrimal apparatus is not the same. Not all of the anatomic factors mentioned necessarily need apply in one patient. The prognosis may rest entirely in the adequacy or inadequacy of the nasolacrimal ostium in the inferior nasal meatus as a drainage aperture. Again, diverticula alone may be the factor or the junction of the lacrimal sac with the nasolacrimal duct may be congenitally of extremely small size, *et cetera*.

The extension of disease processes from the nasal fossa and the paranasal sinuses to the efferent lacrimal passageways is of common occurrence and generally recognized. This is in keeping with the intimate relational anatomy. The incidence of nasal and paranasal disease, however, is greater than secondary disease of the efferent lacrimal apparatus. The extension doubtless is dependent upon the nature, severity and duration of the pathologic processes within the nose and paranasal sinuses, and the degree of intimacy of the topographic interrelations of the several parts (see the nasolacrimal ostium and the paranasal sinuses, *etc.*, referred to before). Of course, minor involvement of the efferent lacrimal channels, secondary to pathologic states within the nasal fossa and the related sinuses may go unrecognized. For example, epiphora may be caused by a mild infection of some portion of the mucosa of the efferent lacrimal channels resulting in congestion of the venous plexus of the lacrimal sac and the nasolacrimal duct and a marked lessening of the lumen. It is difficult to decide in some cases whether the overflow of tears is due to an overproduction or to an inadequate removal. Probably both are factors, since the nasal state may reflexly stimulate the lacrimal gland to greater activity and affect the lumen of the efferent system of ducts by bringing about hyperactivity of the sphincter muscle about the vertical segment of the lacrimal ducts and a reflex engorgement of the venous plexus in the mucosa of the sac and main duct. However, it is well to remember that epiphora is frequently caused reflexly by diseased foci elsewhere and by conditions other than obstruction of the efferent lacrimal channels. Operative pro-



Fig. 11.—Reconstruction of the nasolacrimal passageways of an adult, aged sixty years. Note the regularity of the nasolacrimal duct and the gradual mergence of the lacrimal sac into the nasolacrimal duct at the constriction of the isthmus. $\times 3.2$. Abbreviations as in Fig. 12. (Schaeffer: The Nose and Olfactory Organ.)

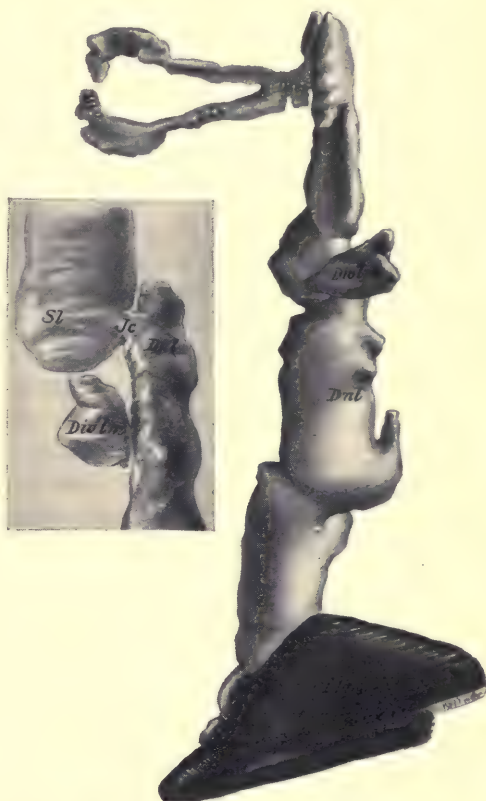


Fig. 12.—Reconstruction of the nasolacrimal passageways of an adult, aged sixty-five years. Especially note the irregularity and diverticula of the nasolacrimal duct.

The inset shows the details of the side-to-side union of the lacrimal sac and the nasolacrimal duct; moreover, illustrates the large bud-like diverticulum from the nasolacrimal duct. $\times 3.2$.

Sl, Saccus lacrimalis; *Dlv*, ductus lacrimalis verticalis; *Dlh*, ductus lacrimalis horizontalis; *Dlc*, ductus lacrimalis communis; *Dnl*, ductus nasolacrimalis; *Mni*, meatus nasi inferior; *Div'lm*, diverticulum of the nasolacrimal duct; *Jc*, junction channel between the lacrimal sac and the nasolacrimal duct.

cedures should be held in abeyance until the case is studied from every angle.

Congenital and acquired strictures, imperforations and other conditions are encountered and the use of the lacrimal probe may be indicated.¹ The lacrimal puncta may need adjustment or enlargement. An endonasal operation upon the nasolacrimal duct or the lacrimal sac may be deemed advisable, the type of operation being dependent upon the kind and position of the obstruction. The only alternative may be the extirpation of the lacrimal sac or even the lacrimal gland. However, the majority of cases of diseased efferent lacrimal passageways require less severe treatment, frequently medical rather than surgical. In directing treatment of the efferent lacrimal apparatus one should always recall a few salient points in the applied anatomy: The mucosa of the lacrimal sac and the nasolacrimal duct between the tunica propria and the periosteum is richly supplied with a venous plexus not unlike an erectile tissue. This stratum is readily engorged directly or indirectly and the resulting swelling of the mucosa is at the expense of the lumen of the sac and duct, especially the latter, since it is encased in a bony canal. The passageways may be of the regular or irregular type. There is no actual stricture or absolute anatomic obliteration of the lumen. The use of stilettes and cannulas in these cases would appear to stimulate reflexly further engorgement, and the good to be derived to be but momentary. The anatomy and physiology are such that the treatment must be directed at the agent which stimulates the receptor neuron in the reflex arc, whether located in the mucosa of the lacrimal passageways, the related nose and paranasal sinuses, or at more remote points.

Again, not infrequently the lumen of the lacrimal sac and the nasolacrimal duct is encroached upon and made irregular by mucosal ledges and partial or complete valves and diverticula. In infection, the swelling and engorgement of the mucosa in these cases result even in a greater obstruction to the flow of tears. As before there is no actual stricture or absolute anatomic obstruction of the lumen.

¹ See S. Lewis Ziegler for valuable contributions to this subject. The Radical Treatment of Lacrimonasal Disease by Rapid Dilatation and Allied Measures, Jour. A. M. A., Vol. 54, 1910. Since submitting copy of this address the writer had the privilege of reading another communication by Dr. Ziegler, "A Further Note on Rapid Dilatation in the Radical Treatment of Lacrimonasal Disease," published in the volume of papers to be presented before the Section on Ophthalmology of the American Medical Association, St. Louis, 1922.

However, the obstruction may be absolute from the physiologic point of view. The advancing lacrimal probe or stilette, doubtless, would lacerate the mucosal ledges and valves and scrape the mucous membrane, which would aggravate conditions. One sees that an impermeable stricture might result from this procedure, and in cases where the primary condition merely was a reduction of the lumen to a potential canal by a swollen and engorged mucosa; that is, a physiologic, but not an anatomic, occlusion.

The majority of specimens show that the lacrimal sac and the nasolacrimal duct are in direct alignment and meet each other by a constriction or isthmus or imperceptibly passing one into the other. A probe is readily passed from the sac into the duct in these cases, unless the isthmus is unduly small.

Occasionally the lacrimal sac and the lacrimal duct are not in alignment and are joined side by side, the lower end of the lacrimal sac extending below the highest point reached by the nasolacrimal duct. In such cases it would be utterly impossible to pass the lacrimal probe from the sac into the duct and if undue force were used the instrument would be pushed through the lower blind end of the lacrimal sac and advance between the nasolacrimal duct and the osseous canal, again to be pushed back into the membranous pathway or to proceed by making a false ostium into the inferior nasal meatus.

It appears certain that the lower aperture of the nasolacrimal duct is a factor in disease and treatment and that it should receive more attention clinically. It may invite infection of the efferent lacrimal apparatus. Again, in a diseased lacrimal duct and sac, the nasolacrimal ostium may be wholly inadequate as a drainage apparatus. Frequently the anatomy is such that a simple operation would result in adequate drainage of the efferent tear ducts. Even troublesome and unexplained epiphora may be caused by a congenitally inadequate type of ostium or aperture.

Heretofore the surgeon did not know what anatomic conformation of the efferent lacrimal passageways confronted him in a specific case. Now that roentgenographic study has been extended to include the efferent lacrimal passageways in the living body, it appears certain that the future treatment and surgery of these structures will be advanced.

I now return to my original thesis—the anatomic type and the variations in the relational anatomy have an important bearing in disease, treatment and prognosis.

THE FACTS AND THEORIES OF COLOR VISION

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I. INTRODUCTION

Guessing at nature's secrets has always been a favorite amusement of the human mind, but in no department of thought has this form of diversion been so wantonly pursued as in the theory of color vision. Only in the domain of metaphysics have the guesses been more wild or untamed by the facts. I cannot hope in the brief time which has been allotted to me to review the multitudinous speculations which have appeared regarding the basis of color vision, and it would be impossible also to discuss in detail the relations existing between even one of these speculations and *all* of the facts in the case. I shall, therefore, confine myself to a more general theme, mentioning particular theories or facts merely by way of illustration. This theme will be to outline the requirements *which must be satisfied by a really acceptable hypothesis*, one which will not be merely a wild, unbridled speculation, but which will seem at home in the company of all of the facts.

The study of color vision leads one immediately into three seemingly separate domains of scientific thought, those of physics, physiology and psychology. Color itself, apart from its stimulus, is now generally conceded by physicists, as well as by philosophers, to be psychologic in character. It is a reality of immediate experience or of the individual consciousness merely. In and for itself color must be studied by the methods of mental science since it proves impossible to identify color either with the wave-lengths of radiant energy or with physiologic activities in the nervous system. However, it is very evident that the knowledge of color vision cannot stop where introspective psychology stops. Color would be *color* if we knew nothing of physiology or of physics, but it would not be *vision*. We must not only recognize the nature and relationships of our visual sensations in and among themselves, but we must understand how they are related psychophysically to the processes of the living nervous system and *via*

these with the factors of the physical environment which surrounds the organism. Thus we are brought face to face with problems not only in psychology but in physiology and physics.

The vast multitude of extant color theories have been propounded by thinkers within all three of the just mentioned domains of science as well as by others whose domains have not been scientific at all. Nearly every available psychologic, physiologic, or physical idea has been utilized. To explain the selectivity of the retinal response, recourse has been had to every known property of radiant energy. The mechanism of the retina itself has been conceived mechanically, chemically, and electrically. Neurologic conceptions of all sorts have been applied or invented. The optic nerve currents have been subdivided or combined, in the course of conduction or at the brain, in a kaleidoscopic variety of hypothetic patterns. Within consciousness itself colors have been classified in many different ways. Nearly a hundred major attempts at an explanation of the phenomena of color vision have appeared in the nineteenth and twentieth centuries. Out of such a host of *trials* we might expect to find something besides *errors*. Our hopes, however, are so poorly fulfilled that it seems pertinent to consider briefly what Mrs. Ladd-Franklin has aptly called the *theory* of color theories.

II. THE GENERAL FACTS OF COLOR VISION

It will be generally conceded that the task of science is restricted to giving an account of what *exists* or what may reasonably be supposed to exist. If, therefore, we should assert that the function of a color theory is to *explain* the facts of color vision, we would be obliged to interpret this as meaning that the theory supplements the facts in the direction of a more *complete* account of the system which is under consideration. An hypothesis concerning the mechanism of color vision cannot legitimately be regarded as being merely a pedagogic or mnemonic device. However much it may assist us in teaching or in remembering the facts of our science, if we are to consider it seriously we must look upon it as a representation of some kind of reality. A scientific hypothesis is an attempt, so to speak, to project the facts beyond themselves, to reach out into the as yet undiscovered domain, to prophesy the facts of the future. In so far as an hypothesis is valid, in so far as it is liable to become in time a fact and not a fancy.

When we say that an hypothesis or a theory should *explain* the known facts, we merely mean that it should combine with these facts

in such a way as to produce a logically coherent account of things. The reason why we need to advance hypotheses lies exactly in the truth that in nearly every department of science the facts which we possess do not in themselves form a coherent logical system. However, the *more* facts we have in any science the more coherent the science becomes, and consequently we believe that a complete understanding of any reality will always yield such a logically satisfactory account. This is why we are justified in using our imaginations to fill in the gaps between the facts, even when these gaps are so large as to leave relatively little indication of the real underlying order which is involved. Our actual data in such a field of investigation as that of color vision are like scraps of a chopped-up picture puzzle, most of the pieces of which have been lost. However, enough may be given so that we have some chance of reconstructing the whole in imagination. It must be realized, however, that the fewer the parts or the facts at hand, the lower is the probability that our reconstruction will be correct.

These considerations should indicate to us clearly both the nature of our problem and the method which we must employ in its attempted solution. A theory of color vision is a picture of reality which must be set harmoniously in the frame of facts. In order to evaluate any such theory we must view it within this frame, and since the frame of facts is a definite thing and the possible pictures many and shifting, we shall do well to study briefly the former before we consider the latter in any detail. In outlining the facts which any theory of color vision must satisfy I shall divide them into two classes, as the *general* and the *special* facts respectively. The general facts comprise the established principles of physics, physiology, psychology and psychophysiology which inevitably bear upon the problem. The special facts consist of the more or less scattered data which have thus far been gathered in the immediate study of color vision itself.

Let us first consider some of the more important of the general principles which are involved in the situation. These principles can be divided systematically into the physical, the psychologic and the psychophysical respectively. Under the first will be included not only the conceptions of physical optics but also those of physiology, since physiology can be classed as a special physical science dealing with the properties of particularly complex material structures called living organisms. The *psychologic* principles relate to experience or consciousness in itself, without reference to any supposed physiologic or

other physical foundations or correlatives which it may possess. The *psychophysical* principles, in turn, involve the *relation* between consciousness and the physical system.

If we look first at the physical side of the problem we find that we have to deal in vision with a mechanism which in its general outlines is capable of being very specifically defined. This mechanism we may designate as that of *visual response*, while recognizing that the meaning which we shall assign to the term response in this connection is somewhat broader than that which is usually given to it. Visual response is one example of a general type of physical process known as *propagation*. Other examples of propagation are to be found in sound, in the conduction of electricity through a wire, or of water through pipes, etc. In each of these processes there is involved a chain of events depending one upon another in serial order, *corresponding events*, or events ultimately traceable to the same initial cause, being displaced successively both in space and in time. In the case of visual response the *initial* event may be conceived to lie in the emission or the reflection of visible radiant energy from a physical object, while the final result may be laid in some adaptive adjustment of the organism to its environment, such as a movement of the eyes or of some other member of the body. Between such initial and final events, and binding them together causally, there lie a considerable number of intermediate *stages*. A schematic analysis of the response from beginning to end would reveal the following successive stages: (1) the object, (2) the visual stimulus, (3) the visual sense-organ process, (4) the retinal or visual-receptor process, (5) the optic nerve *stimulation*, (6) the optic nerve, or afferent nerve conduction, (7) the central or adjustor process, (8) the efferent nerve conduction, (9) the end-plate process, (10) the effector process, (11) the effect.

All of these component processes are linked together as cause to effect, although at the same time each one possesses its own characteristics which make its description different from those of either its cause or its own effects. Thus, the *retinal* reaction is governed by the exact manner in which the radiation is incident upon the retina in forming the retinal image, and yet must be conceived to differ radically from the radiation itself. At the same time we must recognize that those links in the response chain which lie within the organism are ultimately just as *physical* as are those which lie outside of it, the entire system forming a continuous physical mechanism all of which is ultimately reducible to terms of such fundamental units

as electricity and energy. Nowhere within this complex physical apparatus do we find, nor do we need to assume, the existence of such a thing as *sensation*, and hence as *color*. Let us consider briefly some of the known specific characteristics of these several *stages* in the visual response.

The first stage, the visible object, consists of any congeries of electrons and protons constituting a physical body, some of the electrical particles in the surface of which are vibrating at frequencies lying between about 715 and 440 trillion oscillations per second. Such oscillations set up electromagnetic waves of lengths lying between approximately 420 and 680 millimicrons which travel radially away from their sources at the rate of 186,000 miles a second. Before the advent of Einstein we used to believe that these waves were undulations in a medium called the ether, but now we are content to say simply that they are *electromagnetic* in character, which does not prevent them from being waves although they are seemingly deprived of anything in which to undulate. When such waves impinge upon the cornea they are refracted from their normally rectilinear path so that, after suffering further convergence in passing through the crystalline lens, they come to a focus upon the retina to form a pattern roughly similar to that which they possessed at the object. Thus far we are carried very securely by our knowledge of physical optics and the anatomy of the eye, but here our knowledge comes abruptly to a chasm.

The *fourth* stage, the retinal receptor process, is known to exist as a fact but its specific nature is as a fact unknown. Consequently this stage becomes a subject of hypothesis and speculation. Similar statements apply to the fifth stage, or the process by which the retinal excitation is transformed into a nerve impulse. When we come to the sixth stage, however, the conduction along the optic nerve fibers, we are in a somewhat better position. We can detect and accurately measure these nerve currents in animals by means of a galvanometer, and modern studies upon nerve processes in general lead us to very definite conceptions as to the physical nature of such processes. There is no reason to suppose that the human optic nerve currents form exceptions to these generally established principles. The nerve current, as we now conceive it, consists of a series of so-called "all-or-none" pulses, each of which involves a local alteration of the electric polarization and chemic permeability of the membrane surrounding the nerve cell.

We find that the seventh or *central* stage in the response propagation has many representatives in the case of vision, since the transfer of visual nerve currents from sensory to motor channels may take place at a number of different brain levels, and a *succession* of synapses is involved in the transmission of the nerve impulses to the very highest centers in the cerebral cortex. Although we have very little first-hand information concerning the physical nature of the central processes, fairly safe inferences can be made regarding them from a combination of our knowledge of plain nerve trunk conduction, and conduction through entire reflex arcs. The differences between these two types of neural transfer have been properly assigned by Sherrington to the synapse. The four efferent or motor stages in visual response are also quite diverse in separate instances. We must recognize in this connection, not only the motor adjustments of the eyes themselves, highly specific and delicate as these truly are, but also all adaptive movements of any part of the organism which are regulated through vision.

So much for the general frame of physical or physiologic conceptions into which our pictorial hypotheses of the color mechanism must fit. We must now consider briefly another aspect of the frame, the psychologic decorations with which it is embellished, and the manner in which they are associated psychophysically with its more material aspects. The subject-matter of modern psychology is *consciousness and its relations to the physical world*, and consciousness for the modern psychologist is not some sort of subtle relation between a subject and an object, but consists simply in the concrete experiences of human individuals, taken and described exactly as they are found without amplification by the methods of speculative inference and hypothesis which characterize physical thought. Our immediate visual experiences, objective-seeming though they are, comprise the best possible exemplification of what modern psychology means by consciousness. Color, whether chromatic or achromatic, is a psychical element and a psychical element only. It cannot be identified in any respect with its stimulus, radiant energy. The stimulus, according to the physicist, is endowed with wave-length, amplitude and spectral complexity, and it has no color, but the color itself is self-evidently neither long nor wavy, has no amplitude, and is perfectly simple.

Having thus identified color as an essential component of the visual consciousness, we must next consider the manner in which this consciousness is related to visual response. There are two apparent

ways in which we may conceive these two separately defined systems to be associated. One of these ways involves the question as to the place of consciousness in the universe at large, as to the manner in which the physical and the psychologic facts fit together to form a single huge mosaic. This is clearly a philosophic and metaphysical question. The other way is simply that of association through mathematic or logically formal laws. Such laws would tell us how to predict the visual consciousness from a knowledge of the visual response, or *vice versa*, since they would exhibit one of these systems as a function of the other, but they would in themselves offer no explanation of the indicated interdependencies. The metaphysical account, indeed, is really an attempt to explain the psychophysical laws which are established by the empirical methods of laboratory psychology.

Psychophysiology has only begun its task of determining the laws which link the visual consciousness with the mechanism of visual response. However, enough progress has been made to indicate in a skeletal way the principal facts which probably enter into the relationship. It seems quite evident in the first place that although our visual experiences seem to represent the first, or object, stage in the response, their direct determination lies exclusively with the highest central stage or the cerebrocortical activities. Only *via* the dependency of the central stages upon prior and more afferent stages is any relationship established between visual experience and the object or stimulus which initiates the response. If we could reproduce the central activities by artificial means and could then eliminate the entire afferent system, consciousness would remain exactly as before—just as vivid and exactly as objective-seeming.

Ultimate visual psychophysics, then, would involve nothing but consciousness and the brain process, the remainder of the system being purely physiologic or physical. Unfortunately, however, we are still very far from achieving such an ultimate knowledge and we are, therefore, forced to study, in the laboratory or clinic, the relations between the subject's consciousness and stages of the response which are, as a rule, more peripheral than the central activity. In all such cases we are clearly considering *indirect* or mediate psychophysical linkages, and we must realize that all such linkages are subject to physiologic disturbances. It is very probable that visual consciousness actually depends upon activities occurring in some of the very highest association areas of the cerebral cortex, so that even the establishment of correlations between consciousness and the visual pro-

jection area processes does not provide us with direct psychophysical knowledge. The most extensively cultivated department of visual psychophysiology is that of *visual sensation*, which may be defined in terms of the relation of dependency obtaining between the visual consciousness and the first four stages in the response, terminating in the receptor process, but including the environmental forces which govern this process in part.

Although it seems practically certain that the general facts of the psychophysical relationship are as we have described them above, we are nevertheless sadly lacking in exact information concerning the detailed nature of the psychophysical correlations. Consciousness itself is wide open for our examination, and introspective psychology is doing its best to give us a description of its contents. Because of the fact, however, that up to the present time our psychophysical investigations have been largely confined to *indirect* relationships between consciousness and the response activity, we are not in a position to formulate on empirical grounds the equations which connect mind directly with body. Hence the psychophysical connection is another free and fertile field in which to sow the seeds of scientific hypothesis.

It is clear that the rational guesses which comprise a theory of color vision must be consistent with the general principles which we have just considered if they are to be regarded seriously. Any such theory must recognize the extreme complexity of the visual response system and must be prepared to specify the relationships supposed to exist between the elements in the theory and the known subdivisions of the response. Any theory of color vision must deal separately with the data of consciousness and must specify a scheme by which the psychologic color elements are associated with definite factors in the response. Unless the color theorist thus envisages the actual system to which his speculations refer, he has small chance of producing a valid or even a useful sketch.

III. THE SPECIAL FACTS OF COLOR VISION

It appears that a theory or hypothesis of color vision may be regarded as an attempt to fill up the gaps which exist in our *general* knowledge of the psychophysical response mechanisms; and in such a way as to explain or to harmonize with our *special* knowledge of visual psychophysiology. Having briefly reviewed the general facts,

we must now consider succinctly the special visual data, mostly relating to color sensation, which any theory must satisfy.

The first set of special facts which we must recognize are concerned with the nature of color itself. The Colorimetry Committee of the Optical Society of America, in its forthcoming report, will recommend that the term color be used to designate not only visual sensations which manifest hue, such as red, green, blue, etc., but also the achromatic sensations or grays which form a series between black and white. Colors in this sense comprise all possible visual experiences with the exception of the depth factor. Although each discriminable color is in itself perfectly simple, we find it possible to describe any color in terms of *three attributes*, technically known as *hue*, *saturation*, and *brilliance*. The achromatic colors, however, possess no hue and no saturation and are differentiated solely by their differences in brilliance.

The facts regarding visual sensation can be formulated in terms of relations between these three attributes of color and various factors of the response mechanism on the afferent side. We find that characteristic laws connect each of the three attributes with practically every variable which is involved in the response. Ordinarily we associate *brilliance* or apparent brightness with the intensity of the stimulus. We look upon *hue* as an index of wave-length and regard *saturation* as an indication of the purity or homogeneity of the radiant energy forming the stimulus. However, careful studies show that brilliance depends even more fundamentally upon wave-length than upon intensity, the so-called visibility curve expressing the nature of this dependence for daylight vision.

It appears, moreover, that there are two visibility curves, one holding for day vision and the other for night vision, and that these curves represent the responses of two different sets of retinal receptors, the cones and the rods, respectively. The facts which are summarized in the so-called duplicity or rod-cone theory, and which underlie such phenomena as that of Purkinje, must be accepted by any hypothesis of color vision. The relations between brilliance and the spectral constitution of the stimulus are explicable in terms of the two visibility functions and the simple principle of the additive combination of luminosities. Fechner's law, with its upper and lower deviations, provides us with a very accurate expression of the manner in which brilliance, regarded as a psychologic variable, depends upon the

intensity of the stimulus, this law representing not a simple linear, but a quasi-logarithmic relationship.

The brilliance of a color, however, depends not merely upon the three fundamental characteristics of the stimulus, but upon the exact mode of incidence of the stimulus upon the retina, including also the condition of the retina and of the remainder of the visual mechanism at the given moment. Thus the brilliance resulting from a stimulus of fixed inherent character varies with its position in the retinal field, with its form, with its time of application and with the nature of other stimuli which are simultaneously active in outlying portions of the retinal area. Many of these relationships have been worked out quantitatively, but others are only known in a vague way and remain to be investigated in the future. All of them must be explained by a completely satisfactory theory of color vision.

When we pass from the consideration of brilliance or the achromatic factor in vision to the study of hue and saturation we encounter an even more complex system of relationships. The dependency of hue upon the wave-length of the stimulus, in case the latter is monochromatic or homogeneous, has been very definitely determined, but follows a mathematically complex law. Although hue is mainly an index of wave-length constitution, it is also a function of intensity, since at very high intensities all stimuli (of whatever constitution) tend to arouse only yellows or blues, these being of low saturation and passing over at extremely high intensities into a white. The dependency of hue as well as of saturation upon the composition of stimulus mixtures has been very thoroughly studied and the results are represented by the well-known color-mixture triangle. The fundamental fact that in normal vision all of the hues can be reproduced by appropriately proportioned mixtures of three simple homogeneous spectral stimuli must be explained by any color theory which is to merit serious consideration. As part of this set of data we must recognize the importance of the *complementary* relationship between two or more stimuli, by virtue of which they combine in the production of a white. There appear to be an indefinitely large number of pairs of single wave-lengths in the physical spectrum which are capable of combining in the proper intensity ratios to yield this result, but it is of great interest that in the mid-region of the spectrum there is a range of stimuli which have no single complementaries but require the addition of at least two other wave-lengths to yield an achromatic sensation.

Although saturation is ordinarily regarded as an index of the purity or homogeneity of the stimulus, careful measurements show that it varies for different wave-lengths even when they are unadulterated; the mid-region of the spectrum, including the yellow and the yellow green, being much less saturated than the end regions, the blue and the red. As we have already seen, saturation also depends upon intensity, being reduced for all stimuli at extremely high intensities and for certain stimuli which act upon the retinal rods at very low intensities. Both hue and saturation depend, like brilliance, upon the mode of incidence of the stimulus upon the retina and upon the condition of the latter. The dependency of the hue upon the position of the stimulus in the retinal field is summarized in our doctrine of color zones in the visual field which is of much importance in diagnostic technique. Saturation also varies in a parallel manner. The phenomena of color contrast are well known, although they have been studied quantitatively much less than could be desired. The time of action of a stimulus, with the correlated effects of retinal fatigue, influences in a very radical manner both the hue and the saturation which are evoked.

In addition to dependencies in normal persons, illustrated by what we have just said, every theory of color vision must necessarily consider the astonishing way in which color experiences for given stimuli may differ in different individuals. We recognize now that color blindness is only a radical form of a variability which is always to some degree manifest. Not only do hue and saturation vary for given stimulus conditions, but the brilliance which, in our everyday attitudes, we regard as an infallible index of the stimulus intensity also shows radical discrepancies between different observers as indicated by departures of their visibility curves from one another.

Clearly, it would be quite impossible for me to enumerate in this brief address all of the special facts of color vision. My purpose is merely to suggest to you as vividly as I can the extremely involved relationships which are resident in these facts, and thus to show you what a very difficult task it is to construct a really satisfactory hypothesis which will harmonize at once with these special facts and with the more general requirements laid down by physiology and by psychophysics.

IV. THE OLDER THEORIES OF COLOR VISION

In the discussion of color theories all ideas appear to root back ultimately to the hypotheses of Young and of Hering. Both of these theories postulate the existence in the retinal receptors, or somewhere in the afferent section of the response, of three distinct sensitive substances. However, one of these theories is a physicist's doctrine while the other is essentially a psychologist's creation. The Young-Helmholtz hypothesis starts with the stimulus and stops before it reaches consciousness. The Hering scheme, on the other hand, begins with an introspective analysis but becomes vague and unsatisfactory by the time it has been carried back to the stimulus. Physical thinkers are seemingly satisfied with the Young-Helmholtz scheme because they are sensation-blind. Psychologists, for their part, adhere faithfully to the Hering conception because they are stimulus-blind. Needless to say, theories which rest upon a defective recognition of facts, either on the physical or the physiologic side, can scarcely lead to comprehensive explanations.

The Ladd-Franklin hypothesis meets this situation by a well-balanced consideration of both the psychologic and the physical facts in the case. In Mrs. Franklin's theory the three substances become one substance with three specific reactivities. The resulting scheme not only lays due emphasis upon the facts of color-mixture which substantiate the Young-Helmholtz hypothesis, and on the other hand upon the psychologic interrelations of the colors which provide the basis for the Hering theory, but fits better with each of the two sets of facts than did the original doctrines. Mrs. Franklin very cleverly crams two imposing arrays of facts into a single molecule, and thus seems to reduce the data of her science to an ideal logical explanation. As a pedagogic device nothing better than this could be desired, but it may legitimately be questioned whether this very simplicity of the Ladd-Franklin hypothesis does not constitute the principal argument against it, if we are seeking, as we have previously avowed, an actual portrayal of the missing links in the psychophysical mechanism of visual response.

We have seen in our preliminary analysis of the general structure of visual response that the latter mechanism involves a plurality of superposed operations upon the uppermost of which only does color directly depend. All three of the best known color theories, those of Young and Helmholtz, of Hering, and of Ladd-Franklin, are con-

cerned primarily with the mechanisms of a single stage in the response, namely, those of the retinal receptors. Hering, it is true, endeavors to expand the scope of his theory by using the term *Netzhaut* to designate the entire response mechanism from the retina to the brain, but this device does not liberate his theory from the difficulties which go with the simplicity of its assumptions. When we look at the problem in a realistic attitude we see that a theory which deals with only a single stage in the response has very little chance of explaining all of the facts of color vision, and moreover is not consistent with the general demands of the case.

Although by far the greater number of the extant theories of color vision have dealt exclusively with the retinal process there have been in the past an appreciable number of attempts to *subdivide* the visual apparatus, and to hypothecate different mechanisms for the several stages. Theories of this type are known as *zone* theories because they break up the visual mechanism into separate zones. In the most primitive doctrines of this sort only two zones are considered, those corresponding with the retinal and the cerebro-cortical activities, respectively. A plausible suggestion for such a two-zone doctrine would obviously be to assign the mechanism of the Hering theory exclusively to the cortex and to reserve the Young-Helmholtz apparatus for the retina, since, as we have seen, Hering starts with consciousness (which is directly correlated with the central stage) whereas the Young-Helmholtz scheme has been developed mainly with reference to stimuli. A view of this general character has been advocated by von Kries.

Hering, it will be remembered, divides the psychologically primary colors into three pairs of antagonists: black versus white, red versus green, and yellow versus blue. These antagonistic pairs he associates psychophysically with similarly antagonistic processes of catabolism and anabolism in his three visual substances, respectively. He explains a large number of special visual phenomena by means of these antagonistic interactions of the specified color mechanisms. The relationships of complementaries, for example, are said to depend upon the balanced excitation of such mutually neutralizing processes, as do also the processes of simultaneous and successive contrast. If we take the cue from von Kries and place Hering's three substances, or their neurologic equivalents in the brain, we shall naturally look for the explanation of these special particular facts in the central zone of visual response. It seems more probable that antagonistic rela-

tionships of this sort should exist in the nerve centers than that they should be found in the receptors, since the latter are essentially excitatory in their responses whereas in the centers we as often find inhibition as excitation. In other departments of sensation, for example in temperature sensibility, the facts demonstrate immediately the central locus of the antagonistic interactions between simultaneously operative processes. On the other hand, the three substances of the Young-Helmholtz theory seem ideally fitted to operate within the receptor cells of the retina, since their reactions are conceived wholly in terms of excitation and may easily be regarded as instances of a purely physical process of molecular resonance.

Although this dual zone hypothesis excludes any specific consideration of the conduction mechanism which links the retina with the cortex it nevertheless necessitates a set of intermediate assumptions which state exactly how the Young-Helmholtz apparatus in the retina is linked up with the Hering mechanism in the brain. Naturally, we like to conceive of such relations as being of the simplest possible point to point character, but there seems to be no *a priori* reason why we should insist upon such forms of connection. If instead of supposing that the red sensitive substance in the retina is linked exclusively with the red producing elements of the cortex we associate the former with the yellow and white generating brain mechanisms, as well as with the red, we shall obtain a system which has a considerably enhanced capacity for dealing with the special facts of color vision. We shall be able to explain, for example, why it is that a pure spectral red stimulus degenerates into an almost perfect yellow as a result of continued fixation, or adaptation, and we shall be able to account for the differences in luminosity curves which exist between the two common forms of partial color blindness. Thus a combination of the Young-Helmholtz and Hering systems into a two zone hypothesis with a well-considered set of linkages between the two mechanisms should provide us with a theory having a very respectable explanatory power.

Von Kries is by no means the only one among the earlier writers on color vision to suggest a zonal treatment of the visual mechanisms. The very interesting but little known theory of *Donders* is explicitly of the zonal type. *Donders* agrees that the retinal mechanism is as described by Young and Helmholtz, but he supposes that in the brain quite a different apparatus is functioning. This cerebral system consists of a single chemical substance which is capable of being split

along four different diametrical lines of cleavage, corresponding with the four psychologically primary hues, red, yellow, green and blue. Simultaneous cleavage along all possible lines yields the sensation of white or gray, while various mixed modes of cleavage result in colors of intermediate hues or saturation. The theory of Schenck, with its distinction between stimulus-receptors and sensation-stimulators, is also at least implicitly a zone doctrine. G. E. Müller's edition of the Hering theory is an attempt to put the latter scheme into zonal form with a minimum of logical change, while McDougall's discussion of the Young-Helmholtz theory performs a similar service for the latter hypothesis. All of these views are subject to the objection that they are more complex, involving more logically independent assumptions than their parent single zone hypotheses. However, the failure of the latter to satisfy all of the facts in the case indicates that an increased complexity of assumptions is essential in order to provide us with a really satisfactory conception of the mechanisms which are actually involved.

V. SOME NEWER THEORIES OF COLOR VISION

When we see clearly how manifold are the factors which our general knowledge of the visual mechanism inevitably imposes upon our consideration, we must realize definitely how futile must be the search for a simple but all comprehensive explanation of visual phenomena. In the domain of physics it has often proved possible to explain a highly complex array of facts by means of a single simple assumption. General considerations, however, make it highly probable that this possibility is limited to the inorganic realm. While we are committed to the view that organic or vital activities are essentially reducible to the concepts of physics or chemistry this very fact makes it inevitable that living organisms should possess a most elaborate physical structure. Vitality is in essence nothing but intricacy and instability, and we should not be surprised if in biology true explanations should sometimes turn out to be more complicated than are the apparent facts.

If we pursue this line of thought, we shall not only permit our visual theorists to distinguish definitely between the mechanisms which are operative in the retina and those which function in the brain but we shall sanction an even more detailed analysis of the hypothetical mechanisms into zones or stages of the response. On general grounds there appears to be no reason for supposing that the conduction

processes transpiring in the optic nerve fibers are identical, or correlated point to point, with either the retinal excitations or the cerebral processes. Accordingly we might take the further step of introducing a conductional or intermediate zone in our theories, lying between the retina and the brain. This step has actually been taken in a recent very promising hypothesis which is due to the Norwegian psychologist, Harald K. Schjelderup, whose views are worthy of a somewhat detailed presentation.

Schjelderup accepts the classification of colors made by Hering into six primaries, red, yellow, green and blue, black and white, and complies with the usual psychophysical principle in correlating these unit for unit with six corresponding physiologic elements in the brain. These six components in the central zone or stage, however, are conceived to be independently functional mechanisms, not associated together into antagonistic pairs, as in Hering's formula, so that it is possible—as in certain cases of color-blindness—for any one of the cerebral components to drop out without involving others. The admitted antagonism between certain of the colors is laid to the distinctive mechanism of another and more peripheral stage in the response mechanism which Schjelderup calls the *Zwischenprozesse* or "between-process," and which we may conceive to represent the nerve conduction between the retina and the brain. In this zone, Schjelderup hypothecates the existence of paired processes corresponding exactly with the Hering scheme, so that any dropping out of factors here will involve at the least two unitary colors. The individual single color processes of this intermediate zone are conceived to be connected in a simple point to point manner with the corresponding individual processes of the central zone.

Tracing the process backward to the retina, Schjelderup assumes the existence in the receptor zone, of three photochemic substances possessing reactivities to light similar in a general way to those of the Hering hypothesis. One of these substances responds by undergoing oxidation to practically all of the visible wave-lengths of the spectrum. The other two substances, however, are oxidized by the longer waves but are reduced chemically by the shorter ones, the maxima of response either by oxidation or reduction for the two substances lying at different points in the spectrum, so that one reacts mainly to physical red and green whereas the other responds principally to the physical yellow and blue. The linkage between these retinal activities and those of the conduction zone is not, like that between

the latter and the cortical process, a simple point to point correspondence—except in the case of the substance which responds to all of the wave-lengths of the spectrum. This latter substance is linked solely with the intermediate process, which is associated indirectly with the sensation of white. The long-wave oxidation process, however, is linked not only with the red sensation processes of higher zones, but also with those of white and yellow, while the corresponding reduction reaction is associated not merely with the green sensation but likewise with blue and black. The mid-spectral oxidation process, in turn, is connected with both green and yellow sensation processes, while the corresponding reduction activity evokes, through the intermediate mechanism, both red and blue. Although this system seems intricate, it is by no means difficult to apply to the facts and it is probably no more complex than they actually demand. Schjelderup demonstrates that the eight best known forms of color blindness can all be explained in terms of his hypothesis as consequences of the dropping out of one or more of the component activities in his three zone mechanism.

If time permitted I would review for you certain other of the more modern theories of color vision. I would outline for you the recent elaborate speculations of John Joly, in which the modern quantum conception of radiation is brought to bear upon the theory of retinal reaction. I might also summarize the very suggestive speculations of R. A. Houstoun, which take the form of a zone theory. I might refer to the ideas of Barton and Browning, as well as to those of H. E. Ives, bearing upon the physics of retinal response. I might tell you something of Hecht's very suggestive researches regarding the chemistry of visual reactions. I might even refer to the fantastic imaginings of Edridge-Green. I must content myself, however, with the foregoing general comments, which I hope will not leave you altogether without some inkling as to what good a color theory may be, and how good some of the current theories probably are.

THE PRODUCTION AND TRANSMISSION OF CERTAIN EYE DEFECTS¹

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By way of introduction to the discussion of eye defects, I wish to review briefly some points in the embryologic development of the eye. Although it will prove to be an old story to ophthalmologists, I feel, nevertheless, that by so doing I can get before you most effectively the materials I have to present.

Cleavage of the fertilized ovum, formation of the three fundamental germinal layers, and general embryogeny in the rabbit do not differ in any important ways from these same processes in other mammalian forms. Through the successive divisions which begin shortly after penetration of the ovum by the spermatozoon, a mulberry-like mass of cells enclosed by the *zona radiata* is built up. Some of the cells divide more rapidly than others, so that the resulting spherical mass comes to consist of a central group of larger, more granular cells surrounded by a superficial layer of smaller, clearer elements. Soon fluid appears between the central cells and the peripheral layer except at one side. As the liquid accumulates the entire mass becomes transformed into a fluid-filled vesicle consisting of a single layer of small transparent cells with the original central mass projecting from one side into the cavity. The outer layer, termed the *trophoblast*, is concerned only with the establishment of relations between the developing organism and the uterine mucosa. The inner mass is the part out of which the embryo is formed. At this stage the developing ovum is commonly termed the *blastodermic vesicle* or *blastocyst*.

Seen from without, the *germinal area* appears as a circular disc at the upper pole of the blastocyst. Within this disc the cells are rapidly shaping up into the two primitive germinal layers—*ectoderm* and *entoderm*. By unequal growth the disc soon becomes oval, then more or less pear-shaped. At its smaller end a median denser streak, formed by a keel-like thickening of the ectoderm, appears. This is

¹ Illustrated by lantern-slides and living animals.

the so-called *primitive streak*. From each side of the primitive streak cells are budded off between ectoderm and entoderm to form the third germinal layer, the *mesoderm*. The mesoderm grows rapidly until it finally spreads around the entire wall of the blastocyst on the inside. Only that part confined to the germinal area, however, takes part in the formation of the embryo. Ultimately the mesoderm splits into two layers, one of which adheres to the ectoderm and, within the germinal disc, helps to form the body wall; the other adheres to the entoderm, and together, inside the embryo, they form the alimentary tract and its outgrowths.

As the embryo takes form, a shallow median groove, the *neural groove*, appears on its surface in front of the primitive streak. This is the first indication of the central nervous system. The groove is formed by the upgrowth of ectodermal folds, the *neural folds*; by continuation of the upgrowth it is converted into a deep furrow. Ultimately the neural folds fuse above in the mid-axial line and thus form a closed canal, the *neural canal*, which is the forerunner of the cerebrospinal nervous system. The surface ectoderm soon overgrows the neural tube and becomes separated from it. The anterior end of the neural canal enlarges further to form the three primary brain-vesicles, the *fore-brain*, *mid-brain*, and *hind-brain*.

Since it is from the fore-brain that the eyes originate, we shall confine our attention to this region only. It should be understood at the outset that there is a sharp distinction embryologically between the sensory part of the eye and the accessory parts. The sensory part of any sense-organ is always the first to arise in the embryo. The beginnings of the *optic vesicles* appear very early as lateral expansions of the fore-brain, even before the latter is closed dorsally. They are well established in the rabbit embryo by the ninth day. The cavities of the optic vesicles are widely confluent at first with the main cavity of the fore-brain, but as development proceeds this broad communication is rapidly reduced by the downward growth of a ridge of tissue which soon distinctly marks off the optic vesicles from the fore-brain proper by narrowing the original communication to a tubular stalk, the *optic stalk*. Through this stalk, which is attached to the ventral portion of the fore-brain, the cavity of the latter is still continuous with the cavities of the optic vesicles.

The optic vesicles extend laterally until they come in contact with the primitive epidermis or, at least, until separated from it by only a thin layer of connective tissue. Each optic vesicle then becomes

converted into a two-walled optic cup by a process of invagination which occurs at two places, the outer and the ventral surfaces. With the first, the formation of the crystalline lens is associated; the second constitutes the so-called *choroidal fissure*. The inner wall of the optic cup, destined to become the sensory part of the retina, is much thicker than the outer wall, which will eventually become the pigment layer of the retina. These infoldings of the walls of the optic vesicles to form the optic cups commence on the tenth day in the rabbit and are completed by the fourteenth day. The crystalline lens is making its appearance simultaneously with these changes.

In the formation of the choroidal fissure the infolding extends a certain distance along the optic stalk toward the brain, so that, near the eye, the optic stalk is a tube deeply grooved along the under surface by an infolding of its thick lower wall. It is along this groove, through the choroidal fissure, that the central artery of the retina gains admittance to the eye. This artery supplies the retina throughout life, but during the development of the eye it also supplies the vitreous body and the capsule of the growing lens. At first rather wide, the choroid fissure becomes more and more narrow until under normal conditions it closes altogether soon after the entrance of the arterial blood-vessel, but, as you well know, this fissure may remain unclosed, producing the not uncommon malformation known as *coloboma*. I stress this condition, at this point, because of its bearing upon some of the eye-anomalies I shall describe later.

The lens first appears before the invagination of the primary optic vesicle, at the place where the epidermis passes over the surface of the latter. At first a slightly thickened disc, it soon invaginates to form a small pit, which by approximation of the edges becomes converted into the *lens-vesicle*. In the cavity of the lens of the rabbit and of man epithelial cells occur; but their significance is unknown. They ultimately degenerate and disappear. The lens-vesicle finally becomes constricted off from the surface-epidermis.

As the optic cup invaginates it and the lens draw apart somewhat although they remain connected by protoplasmic strands. The inner wall of the lens-vesicle very early increases in thickness and encroaches on the lens-cavity but the outer wall remains thin.

This thin anterior layer persists throughout life as a simple layer of cubical cells, the so-called *lens-epithelium*; but the cells of the posterior layer grow forward into the cavity as the lens-fibers. The central fibers are straight and long; the peripheral fibers, somewhat

curved, become shorter and shorter toward the surface of the lens, where they merge into columnar cells which are continuous with the anterior lens-epithelium. The cavity is gradually obliterated and the developing lens thus becomes a solid body. The part formed so far, however, constitutes only the core of the adult lens. Around this core (beneath the lens epithelium of the front surface of the organ) successive coats of lens-fibers arise, lying in layers one over another like the coats of an onion. These secondary fibers arise at the equator of the lens, and, increasing rapidly in length, spread over the ends of the first-formed fibers.

In discussing the formation of the optic cup it has already been pointed out that the deeper parts of this structure become the retina. There is a thinner outer rim, however, marked off from the retinal region by the so-called *ora serrata*. This thinner portion, together with invading mesenchymatous tissue, is concerned in the formation of the *iris* and the *ciliary body*.

The *cornea* and its associated structures are formed mainly by a layer of invading mesenchymatous tissue which surrounds the lens and separates it by a thin sheet from the epidermis. This sheet thickens by immigration of adjacent cells; then it separates into two layers, one of which, the *pupillary membrane*, remains thin and becomes very vascular, while the other becomes the early cornea; covered, however, by a corneal epithelium of ectodermal origin. Between the two layers the anterior chamber of the eye appears as a narrow fissure which becomes filled with fluid, the *aqueous humor*, and gradually increases in size with the appearance of the iris. In mammals, the pupil is closed at first by a thin vascular connective-tissue membrane (pupillary membrane) which also forms the anterior part of the *tunica vasculosa lentis*. The blood-vessels of this anterior part are continuous with those of the connective tissue at the margins of the optic cup. It will be noted from this that during its main period of development the lens of the fetal rabbit is surrounded by a membrane, the posterior part of which is supplied by the hyaloid artery, the anterior surface, by branches of the anterior ciliary arteries. Thus, by the thirteenth day, the lens is relatively large and is inclosed in a membrane richly supplied with blood-vessels. Before birth the blood-vessels of this membrane disappear and the membrane becomes (or merges with?) the lens-capsule.

During its earlier formation the cavity of the optic cup is practically filled with the newly forming lens, but as development proceeds the

retinal layer of the cup draws away from the lens and thus forms the vitreous chamber. However, nuclear-free protoplasmic threads still connect the lens and the future retinal epithelium, and it is this network which apparently constitutes the formed elements of the *vitreous body* and the *zonule of Zinn*. There is also some invasion by mesenchymatous tissue. The vitreous matrix rapidly increases in amount and becomes more and more watery. Later it is surrounded by a structureless membrane, the *membrana hyaloidea*. As noted earlier, the *hyaloid artery*, a branch of the retinal artery, extends across the vitreous chamber from the entrance of the optic stalk and not only supplies the *tunica vasculosa* of the lens but gives off branches to the vitreous body itself. Along with the hyaloid artery these disappear before birth, leaving the vitreous body of the adult destitute of blood-vessels. The old path of the hyaloid artery persists, however, as a canal filled with fluid, the *canalis hyaloideus*, or canal of Stilling.

The *optic nerves* arise in connection with the original optic stalks. The interior of each stalk becomes converted into a glial network through which nerve-fibers having their origin from ganglion cells in the retina grow inward to the brain.

The *sclerotic* and *choroid* coats respectively arise as two layers differentiated from the mass of mesenchyme which surrounds the optic cup. In front, the cornea is continuous with the sclerotic. The two are at first identical in structure; it is only later that the cornea becomes transparent. In the rabbit the sclerotic coat becomes glistening white. It contains no cartilage.

As we have seen, the retinal part of the eye has a very different origin embryologically from the outer and middle coats. It never becomes closely fused with them and may thus easily be detached. This is a condition which has direct bearing upon certain of the eye-anomalies we have found, characterized by detachment of the retina.

A number of interesting facts about the developmental mechanics of the eye have come to light through the efforts of experimental embryologists. It will not be time misspent, therefore, if we digress a bit to review some of the more significant of these, which have bearings on our problems of eye-anomalies. For example, there has been much discussion regarding the way in which cyclopean defects occur. The condition, as you recall, ranges all the way from an aborted or a full-sized single, median eye, through eyes showing varying degrees of doubleness to two separate eyes that are merely unusually close together. While *cyclopia* has long been known to medical men as a

rather rare teratological phenomenon, experimental embryologists, and notably among them Stockard (1909), have developed chemical means by which, in certain species of fish, at least, cyclopia can be induced almost at will. Stockard has found that when the developing eggs of *Fundulus heteroclitus*, for instance, are subjected to the action of various magnesium salts dissolved in sea water, a large percentage of them—as many as sixty in one hundred individuals—develop a single median eye instead of the ordinary pair. He and others have also secured cyclopean monsters through the use of alcohol, ether, and certain other reagents.

Stockard believes such results are due to an early developmental arrest, and he raises the question as to the original location of the optic elements in the embryonal central nervous system. Visibly, the eye-antecedents first appear as paired outgrowths from the primary fore-brain, but what is their earlier condition in the medullary tissues? Stockard (1913), partly as an inference from the way his cyclopean monsters are formed, but also from the outcome of certain operations he performed on embryos of one of the salamanders (*Amblystoma punctatum*), has come to the conclusion that the original material of the optic outgrowths is median and single, and that later it separates into two growth-regions which, developing in lateral directions, give rise to the primitive optic vesicles. He regards *cyclopia* as due to an arrest in eye-formation while the optic-elements are still in the median axis of the developing nervous system. This is contrary to the older view which located the antecedents of the eyes in lateral positions along the margins of the medullary plate, and held that the cyclopean defect was due to a coming-together and fusion of the incipient optic vesicles in the median plane.

Stockard submitted the problem to experimental test by excising various parts of the medullary plate. He found that in the very young embryo, operations which removed the tissue of the median line prevented the development of eyes, while operations in which the removed tissue was confined to a lateral position though still involving the lateral fold, in nine of the eleven embryos employed, did not prevent the development of a perfect eye on the operated side as well as on the other side. His inference is, of course, that the material for both eyes was medially located at the time of operation. Lateral operations upon somewhat older embryos indicated that the eye-antecedents, in these later stages, were coming to occupy more lateral regions. Apparently the different degrees and kinds of cyclopean

defect depend in large measure upon the stage in development at which the arrest occurs. If cyclopia is complete, according to Stockard's theory, obviously the arrest must come before the median tissue has separated into two lateral components. Stockard's comparative study of cyclopean individuals in which the single eye grades all the way down from an eye of normal size to one of extremely minute dimensions confirms him in the belief that the earliest eye material occupies a median position. Differences of size are due to different degrees of completeness of the arrest. The "hour-glass" eye, or incomplete cyclopia, he regards as due to a later or a less complete arrest. To secure cyclopia in *Fundulus* the embryo must be subjected to appropriate stimuli not later than the fifteen-hour stage of development. Since the optic vesicles do not appear under normal conditions until about thirty hours of development, it is apparent that important steps in their formation are in progress long before the two vesicles themselves become visible.

Under the same experimental condition which causes cyclopia an entirely different type of eye-anomaly is common, in which one normal eye occurs in the usual lateral position while the eye of the opposite side may be wholly lacking or may show various degrees of imperfection. Such an anomaly is termed *monophthalmia asymmetrica*, to distinguish it from the cyclopean type. Stockard is inclined to believe that in such cases some inhibitive influence becomes operative on the one center of growth after the future eye centers have begun to be localized in more or less lateral positions. He found some notable examples of this in chick embryos from eggs which had been exposed to alcohol fumes, as well as among his fish embryos. Under such methods of treatment not infrequently, also, both eyes would remain small and defective. Inasmuch as the same types of defect may be produced by any one of a number of different chemical or physical means, it is clear that the response in such cases is not specific with respect to a given agent. The widely differing agents apparently merely act similarly on the embryonic organism or on certain of its parts at critical stages in their development. Stockard's explanation of such defects as developmental arrests of particular structures at such critical periods is the most acceptable that has been offered.

Child (1915, 1921) has shown in numerous studies extending over a period of twenty years or more, on a wide range of animals, that the developing embryo of bilaterally symmetric forms has a marked polarity along an antero-posterior axis, with different rates of metab-

olism at different points along the axis. In the early embryo the developing head-end always shows the highest rate of activity, the activity gradually diminishing toward the posterior end. Child has also shown that the more active a region is, the more susceptible it is to adverse chemical or physical influences. Since the rate of oxidation gradually diminishes along the gradient from the region of highest activity, he holds that differences in oxygen supply probably play a very important part in the local metabolic differences.

In a recent experimental study on twins, double monsters and other deformities, and on interactions among embryonic organs, Stockard (1921) likewise attributes much importance to reduction of oxygen supply at critical stages of embryonal or organal development in causing arrests which result in the production of abnormalities. He shows that by temporarily lowering the temperature and thereby reducing the rate of oxidation, or by directly cutting off the supply of oxygen, the normal, continuous course of development of the embryo or of some embryonic part may be interrupted, with the result that characteristic suppressions or distortions may occur. Interruption of development during late cleavage, for example, results in the production of a considerable number of twins and double individuals.

During the past twenty years or so experimental embryologists have also furnished us with various interesting and significant facts concerning the origin and development of the early crystalline lens. There has grown up through their efforts, in fact, a distinct "lens-problem." As we have already seen, under normal conditions the lens develops in very close association with the optic cup. The question arose, therefore, as to whether the lens, like the optic cup, was in a sense a self-differentiating structure, or whether it owed its occurrence to a contact-stimulus exerted by the optic vesicle upon the overlying ectoderm. The belief that the ectoderm was unable to give rise to a lens without the optic vesicle stimulus was experimentally supported by the early work of Spemann (1901, 1903) and Herbst (1901). Spemann, for instance, showed that injury to the medullary plate of the frog (*Rana fusca*) might inhibit development of the optic cup, or, if the cup developed, might keep it from coming into contact with the ectoderm. In either event no lens formed. If, however, in spite of the injury, the cup or even a part of it reaches the epidermis, a lens forms at the point of contact. Lewis (1904, 1907a, 1907b) also, from a series of studies on other species of frog and on the salamander (*Amblystoma*), concluded that lens would not arise from the normal

lens-forming region of the ectoderm without the contact-stimulus of the optic vesicle on the inner layer of the ectoderm. He showed, moreover, that a piece of ectoderm taken from another part of the body and grafted over the optic cup would form a lens, and also that the optic cup experimentally removed and engrafted under the epidermis of another region of the head would stimulate the development of a lens at any point of the surface-ectoderm with which it came in contact. Lewis found, furthermore, that the optic cup of one species of larval amphibian can probably even stimulate the formation of lens from the ectoderm of another species. Thus, optic vesicles of the frog, *Rana sylvatica*, were transplanted beneath the head-ectoderm of salamander embryos (*Amblystoma*). Lenses were later found associated with several of the transplanted eyes. He was not absolutely sure of the source of these lenses, but as the amblystoma embryos had normal lenses in their own eyes, uninfluenced by these secondary lenses, the inference was that formation of the latter had been initiated by the transplanted alien optic vesicles. Spemann reports having secured lens-formation in the ventro-abdominal ectoderm of *Bombinator*, by transplanting it over the bared optic vesicle of *Rana*.

From the various experiments just related it is obvious that epidermis that does not normally give rise to lens will differentiate into a lens if brought into contact with the optic vesicle. It would seem that there are no special cells predetermined to form lens. In his later papers, however, Spemann (1908, 1912) modifies his opinion somewhat on the basis of other experiments, concluding that while in some species of frogs lens cannot develop if the optic vesicle fails to make contact with the overlying epidermis, in at least one species the epidermis is capable of engendering lenses without such contact. King (1905) and Stockard (1910), furthermore, maintain on the basis of their experimental work that there can be independent origin and development of the crystalline lens.

In his studies of the problem, Stockard (1910) substituted chemical means of experimentation for the mechanical operations employed by other investigators. The developing eggs of the minnow, *Fundulus heteroclitus*, were subjected for a time to the action of magnesium salts, alcohol or ether. Such treatment frequently prevented the outgrowth of the optic vesicles. In this way he secured embryos with the optic outgrowths as entirely lacking as in specimens with these parts cut out. This method has the advantage of avoiding the injury to the ectoderm which results from mechanical operation. In many of the

fish embryos with optic outgrowths thus suppressed by chemical means, he found developing crystalline lenses. He concluded, therefore, that typical lenses may originate and develop from ectoderm without any direct stimulus from optic vesicle or cup. The possibility that optic vesicles may have arisen, stimulated the formation of lens and then degenerated, he believed, judging from the hundreds of *Fundulus* embryos he has studied, is entirely out of the question.

Stockard (1910b) takes the ground, therefore, that while unquestionably an optic vesicle or cup possesses power to induce lens-formation from various regions of the ectoderm with which it may come in contact, there can also be independent formation of the lens. He believes that independent lens-forming power is present over the entire head, but that it diminishes from before backward until trunk-ectoderm is reached, which no longer possesses this capacity. He maintains that when, in eyeless larvæ, free lenses occur, they usually arise near the anterior tip of the head. Posterior lenses as large as anterior ones may appear, but they occur less frequently. He thinks that the ectoderm of the head has a distinct tendency to form lens but that it exercises this capacity much more certainly and effectively when stimulated by optic vesicle or optic cup.

In a later critique of the whole lens problem Werber (1918) argues that the original interpretation which regarded actual contact of optic outgrowth with the ectoderm as essential to lens formation is the correct one. In experiments based on extirpation of the optic vesicles or optic cups, for example, he maintains, from evidence he finds in his own studies (1916c), that fragmentation of the optic vesicle has occurred, so that bits of it sufficient to stimulate lens-formation have been left behind and he thinks that the same thing has probably happened in the operations of others. In teratological material, likewise, he points out, there is frequently much dissociation and shifting of tissues. He cites a case in his own experience in which the optic cup became profoundly dissociated and scattered so that fragments of it came into contact with the ectoderm of the head in many places, with the result that lentoid bodies were formed in great numbers.

In the face of the amount of evidence brought forward by Stockard, however, Werber's arguments against independent lens-formation are not very convincing, at least, as regards the cases of chemically induced monstrosities in *Fundulus*. The conclusion that ordinarily the lens arises only in response to a stimulus from the optic vesicle,

but that under certain conditions it can arise independently seems to be the most justifiable one in the present state of the evidence.

Experiments have shown that lens-formation may also be initiated in or by later derivatives of the optic cup. Thus the epithelium of the iris can apparently undergo lens-formation, as can also the retinal cells. For example, when the lens is removed in certain amphibia, a new lens may be generated from the margin of the iris. Wachs (1914) found, moreover, that when he implanted a fragment of iris in the vitreous chamber of an eye deprived of its lens, the implanted fragment and the animal's own iris each formed a new lens. It has also been shown experimentally that retinal cells can both furnish the stimulus for lens-formation and respond to it, since retinal lentoids can be induced to form by experimental injury of the retina.

It is an interesting fact that the mechanical effect of contact may play an important part in lens "regeneration." Fischel (1902), for example, removed the lens of a salamander and replaced it by a small spherical bit of potato. When this was large enough to fit the pupil, new lens-formation did not take place, but if the diameter of the foreign body was smaller than that of the pupil, an attempt toward new lens-formation occurred. More recently Fischel (1916) found, in larvæ of the salamander (*Salamandra maculosa*), when the extirpated lens was transplanted under the skin of various parts of the head or trunk, that while the lens degenerated more or less and tended to be resorbed, the overlying skin underwent changes which eventually led to its becoming transparent and very similar to a cornea. Wachs (1914) had earlier obtained much the same result with a transplanted fragment of the optic cup containing both iris and retina. Such an effect, however, is probably mechanical and not adaptive, as would appear at first sight, since Cole (1922) has shown that although tail-skin grafts over the eyes of frog-tadpoles become absorbed in such a manner as to tend to expose the eyes and restore vision, absorption also occurs in tail-skin grafts over small hemispheres of glass or celloidin. Cole regards such absorption, therefore, not as functional regulation, but as entirely a mechanical reaction to the tension caused by the curvature of the underlying object.

As work in the field of immunology has progressed during the past quarter of a century it has become increasingly evident that the serologic reactions all have their broader biologic aspects. They must in last analysis be but special manifestations of the general processes which underlie all life phenomena. Unquestionably the

serologist has put important tools and ideas into the possession of the experimental biologist which may be utilized in new attacks upon certain fundamental biological problems.

The hemolysins, for example, discovered by Bordet in 1895, are now known to be special members of a general class of substances termed cytotoxins or cytolsins. For just as alien red blood-cells lead to the production of specific hemolysins, so various other materials, as leukocytes, nervous tissues, spermatozoa and crystalline lens—any foreign protein, in fact—when injected into the blood-stream of an unrelated species, will cause the formation of lytic substances more or less specific for the antigen used in the immunizing process. All cytolytic sera so far studied have been found to be more or less hemolytic, and it is probable that none acts exclusively upon its own antigen. While a particular cytolytic serum may affect some other tissues, it attacks the special tissue used as antigen much more vigorously.

Although presumably distinct from one another, the various classes of the so-called *antibodies*—precipitins, agglutinins, bacteriolysins, cytolsins or cytotoxins, etc.—seem to have many points of similarity, as, for instance, their method of origin, their reaction to heat, and, in some cases, their mode of operation. Chemically their natures are still unknown. Considerable evidence of their close association in some way with the euglobulin constituent of the blood is appearing in various recent researches.

To the biologist viewing this fascinating field, many questions arise. If, for example, it is possible to originate in living organisms antibodies which will destroy particular tissue-elements, is it not possible to secure similar selective action on certain parts of the developing embryo? May not serologic methods enable us to make a new attack upon the long-standing problem of the inheritance of somatic modifications, or that of provoking specific modifications in the germ through direct operation of external agents? If a special serum can be developed which will single out and destroy a certain element of the adult, is it not possible that there is sufficient constitutional identity between the mature substance of such a part and one or the other of its material antecedents in the germ, that the latter may also be influenced specifically by the serum in question? If external influences *can* be transmitted to the germ-cell, it is clear that in higher animals the one obvious means of conveyance is the blood.

In an attempt to find answers to certain questions of this kind I and my research associate, Dr. E. A. Smith, began various experi-

ments some six years ago which we are still continuing. Among other things we undertook, by means of cytolsins, to produce antenatal effects in fetuses. Our main work in this direction has been on rabbits with fowl-serum immunized against rabbit-lens, although we have also experimented somewhat with mice and with guinea-pigs. I shall confine my discussion largely to certain eye-abnormalities we secured in fetal rabbits, and to the inheritance of such defects.

In our first experiments¹ the lenses of newly killed young rabbits were pulped thoroughly in a mortar and diluted with normal salt solution. About four cubic centimeters of this emulsion was then injected intravenously or intraperitoneally into each of several fowls. Four or five weekly treatments with such lens-emulsions were given. A week or ten days after the last injection the blood-serum of the fowls was ready for use. The rabbits had been so bred as to have their young advanced to about the tenth day of pregnancy, since from the tenth to the thirteenth day seems to be a particularly important period in the development of the lens. As we saw in reviewing the embryology of the eye, the lens is then growing rapidly and is surrounded by a rich vascular network that later disappears. From four to seven cubic centimeters of the immunized fowl-serum were injected intravenously into the pregnant rabbits at intervals of two or three days for from ten days to two weeks. A number of the rabbits died from the treatment and many young were killed in utero. Of sixty-one surviving young from mothers thus treated, four had one or both eyes conspicuously defective and five others had eyes that were clearly abnormal. It is possible that still others were more or less affected, as we judged only by conditions easily visible. In some of the descendants of this stock, indeed, ophthalmologists who have examined the eyes more thoroughly have pointed out defects which we had overlooked, and occasionally rabbits, that in their earlier months passed for normal, have later manifested defects in the lens or in other parts of the eye.

The commonest abnormality seen in both the original subjects and in their numerous descendants was partial or complete opacity of the lens (Plate I, Fig. 4), usually accompanied by reduction in size of the eye (Plate I, Fig. 2). In a few of our later strains in a different experiment, however, we have had several cases of enlargement of the eye, or *buphthalmia* (Plate I, Fig. 3). Among the rabbits I brought with me for demonstration there is one of this type which I shall be glad

¹ Guyer and Smith, 1918, 1920.

to have you examine. Other common defects which have appeared are cleft-iris, displacement of the lens, persistent hyaloid artery, bluish or silvery color instead of the characteristic pink of the albino eye, microphthalmia, and even almost complete disappearance of the eyeball. The cases of cleft-iris, or *coloboma*, range all the way from a narrow slit in the lower edge of the iris to a broad wedge- or U-shaped opening which amounts practically to the absence of the entire lower part of the iris. The cleft may be confined to the iris or it may extend back deeper into the eye. When one takes into account the early embryology of the eye, it is easy to see how such clefts result from failure of the choroidal fissure to close as it should do normally. The bluish or silvery color, I am told by ophthalmologists who have examined the rabbits, is due mainly to detachment of the retina. Here again, when one recalls the loose embryologic connection between the retinal layers of the eye and the outer coats, even in the normal eye, it is easy to see how almost any distortion of the eyeball, unevenness of growth, or accumulation of fluid might bring about such detachment.

Many of the eyes take abnormal postures (Plate I, Fig. 3). This is particularly true in some of our later strains. One or both eyes are likely to be strongly rotated downward or backward. The backward-rotation is carried to such an extreme in some cases that the cornea is visible only when the eyelids are drawn back at the outer corner (Plate I, Fig. 3), or occasionally when the animal attempts to roll its eyeball forward. In such eyes the exposed sclera in front usually bulges (*staphyloma*) and becomes transparent, simulating a cornea. When we first came across this anomaly, in fact, we thought that we had a rabbit with a double eye on each side. I have brought one such individual with me for demonstration.

Taking into account the method of embryologic development—the relations of lens, optic cup and choroidal fissure—the defects, except those of the muscular attachment, are practically all such as might reasonably be attributed to arrests of development based upon early lens-defect. It is possible, to be sure, that we have developed antibodies against other eye-tissues as well as against the lens, since undoubtedly more or less of the aqueous humor and the vitreous body adhered to the lenses when we removed and pulped them for the original injections. Moreover, if proteins from other parts of the eye are ever in solution in the humors, they too may have been present in the antigen. Each individual protein, of course, has the capacity

PLATE I



Fig. 1

Fig. 1.—Showing appearance of normal eye.



Fig. 2

Fig. 2.—Microphthalmic eye with cleft iris and opaque lens; eyeball rotated downward somewhat.



Fig. 3

Fig. 3.—Buphthalmic eye with staphylomatous sclera. The eyeball is so rotated backward that the edge of the cornea is just visible at the upper outer angle of the lids; the lenses in both eyes are opaque.

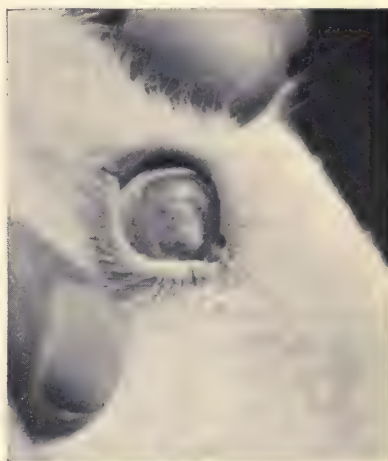


Fig. 4

Fig. 4.—Showing opaque lens and coloboma of the iris.

for engendering antibodies specific for itself. Even the lens is composed of at least four proteins: albuminoid (constituting the lens-fibers), alpha-crystallin, beta-crystallin and albumin. According to Jess and Reiss (Jess, 1920), in their study of the chemical changes which take place in cataract, alpha- and beta-crystallin, both soluble in water, make up the greater part of the lens of the young animal. These gradually decrease in quantity with age, accompanied by sclerosis—a process even more in evidence in cataractous lenses.

In some of our animals we find that an eye defective at birth, particularly if microphthalmic, may undergo further degeneration, characterized by collapse of the eyeball and resorption, so that the eyeball may eventually disappear entirely. The eyes of the mothers originally injected have always remained apparently unaffected. This is probably due to the fact that the lens-tissue of the adult is largely avascular, and that, therefore, the injected antibodies did not come into contact with it.

That the changes in the eyes of the fetuses resulted from the specific action of lens-antibodies is indicated by the fact that in the original experiment, in not one of the forty-eight controls obtained from mothers which had been treated with pure fowl-serum or with fowl-serum immunized to rabbit-tissues other than lens, was there any evidence of eye-defects. I may add that since then, among over five hundred young obtained from mothers which are being experimented upon for other purposes with various types of sera or protein-extracts, or with typhoid bacilli, just before or during pregnancy, not a single case of eye-defect has appeared. To one familiar with the results obtained by the experimental embryologist, which show how susceptible the eye is in early embryogeny to any kind of harmful influence, the natural inclination is to regard such abnormalities as due merely to a general poisonous or inhibitive effect, rather than to specific antibodies in the blood-serum. That lens-defects may be produced by general chemical or physical means is undeniable. I know of no case yet, however, where they have become inheritable. Bagg (1922), for example, has recently found that as a result of exposure of rats to radium emanation (gamma-ray radiation) during late pregnancy, some of the young, after birth, developed eye-defects. In his paper he gives photographs of an adult in which both lenses have become opaque and the left eyelids nearly closed. As a rule, such fetally irradiated young showed other marked defects, particularly of the nervous system, and were usually sterile.

Regarding our own rabbits I can only repeat that we have never obtained the defects in question except with serum carrying specific antibodies. In any event, should the effect have originally been a general rather than a specific one, it is obvious that, germinally considered, it must sooner or later have become specific, since the anomalous eye-condition appears generation after generation without any recognizable accompanying malformations of other parts of the body.

Before passing on to the question of inheritance, I may say that by way of control, for genetical studies, in addition to what we have termed our 3A1 line, we developed another line from wholly unrelated stock, our so-called 16A1 line. Moreover, we have established still a third strain, the 84 line, which was started, not by means of fowl-serum immunized to rabbit-lens, but by the use of pulped rabbit-lens intravenously injected directly into rabbits just before or during their pregnancy. In this last case the rabbit must herself have developed antibodies against the invading lens-material. Out of eleven different females so treated, in twenty-three matings, only one individual gave us young with abnormal eyes. These defects are of the same general nature as those secured by means of fowl-serum immunized to rabbit-lens, and they behave similarly in inheritance.

As already indicated, once the defect is secured, it may be transmitted to subsequent generations through breeding (Fig. 1). So far, in the 3A1 line, we have succeeded in passing it down through nine generations. There is no reason apparent why it will not go on indefinitely, since the imperfections tend to become worse in successive generations, and also to occur in a proportionately greater number of young. The same genetical conditions hold for the other lines, although because of their more recent origin, we have manifestly not been able to carry them through so many generations.

The transmission is not infrequently of an irregular unilateral type (Fig. 1), sometimes only the right, at others only the left, eyes showing the defect. In this respect it resembles genetically such anomalies as brachydactyly or polydactyly in man. In later generations there has been an increasing number of young with both eyes affected.

Though not analyzed completely as to its exact mode of inheritance, the abnormal condition has in general the characteristics of a Mendelian recessive. When either defective-eyed males or females are bred to normal-eyed individuals from other strains, for instance, only normal-eyed progeny result in the first generation, but the abnormal condition may be made to reappear in subsequent generations if

appropriate matings are made. If we were dealing with a pair of simple Mendelian characters, the young from two individuals with

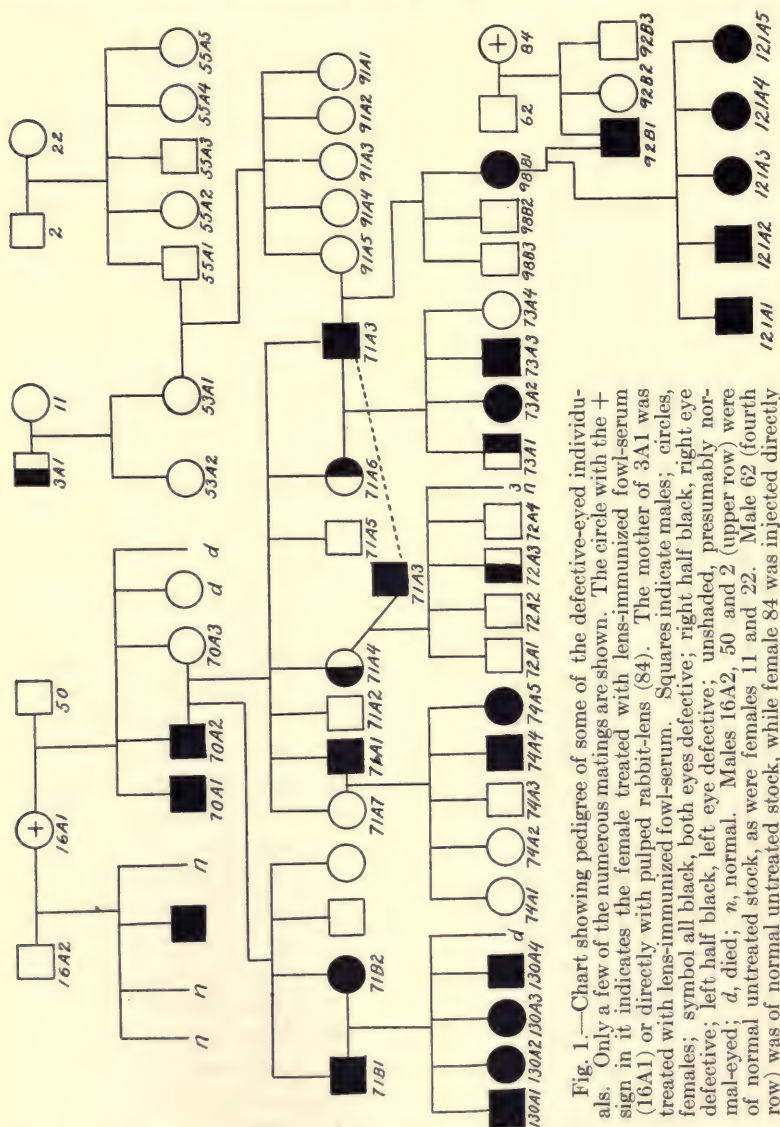


Fig. 1.—Chart showing pedigree of some of the defective-eyed individuals. Only a few of the numerous matings are shown. The circle with the + sign in it indicates the female treated with lens-immunized fowl-serum (16A1) or directly with pulped rabbit-lens (84). The mother of 3A1 was treated with lens-immunized fowl-serum. Squares indicate males; circles, females; symbol all black, both eyes defective; right half black, right eye defective; left half black, left eye defective; unshaded, presumably normal-eyed; *d*, died; *n*, normal. Males 16A2, 50 and 2 (upper row) were of normal untreated stock, as were females 11 and 22. Male 62 (fourth row) was of normal untreated stock, while female 84 was injected directly with pulped lens.

the same recessive trait should all show this trait. Two of our defective-eyed rabbits, however, when bred together, are likely to

produce some normal-eyed young. If, therefore, this inheritance is to be interpreted in terms of Mendelism, there is probably more than one pair of unit-factors involved.

To meet the objection that we are not getting instances of true inheritance but merely placental transmissions of antibodies or related substances from the blood-stream of the mother in each successive generation, we have established the descent through the male line in

Male Line Extractions

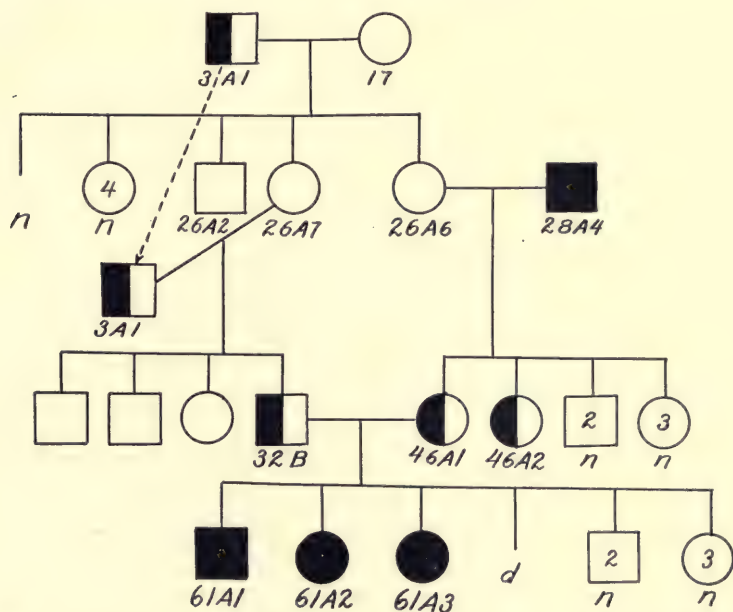


Fig. 2.—Inheritance of the defects through the male line. It is plain that individuals of the 32B, 46A and 61A series could have derived their defects only from male ancestry originally, since female 17 was of normal and unrelated stock. Symbols same as in Fig. 1.

a number of cases, one of which is represented in Fig. 2. To do this, females from strains of rabbits unrelated to our treated stock were mated to defective-eyed males. The first generation produced in this way was invariably normal-eyed; that is, the defective condition was recessive to normal condition. When, however, females of this generation were mated to defective-eyed males, or to normal-eyed males of similar derivation to themselves, the defects reappeared in

some of the progeny, somewhat after the manner of an extracted Mendelian recessive. It is obvious that the normal condition could have been introduced into these new strains only through the germ-cells of the males, and that its transmission is, therefore, an example of true inheritance.

I feel that in establishing and developing from unrelated stock three different strains of defective-eyed rabbits—two (3A1 line and 16A1 line) by the use of fowl-serum immunized to rabbit-lens, the other (84 line) by direct injection of rabbit-lens into a pregnant rabbit—we have placed our results beyond the bounds of coincidence or chance. We can also cite further the production recently of similar lens-defect in the young of the guinea-pig, if need be, although we are not yet ready to report on this latter series of experiments.

To the biologist, perhaps the most interesting fact brought to light in these researches is the possibility of directly or indirectly inducing germinal changes by means of antibodies developed in an animal's own body against tissues taken from individuals of the same species. Such a result together with another I have obtained in inducing the male rabbit to develop spermatotoxins against its own spermatozoa (Guyer, 1922a), lend support to the idea that an animal can build antibodies against its own tissues when these are misplaced, altered or injured, and that such antibodies may so affect the germ-cells as to induce germinal changes. Since I have discussed this point rather fully in recent papers (Guyer, 1921, 1922b, 1922c), I need not enter into it here.

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A NEW TECHNIC FOR THE APPLICATION OF THE METHOD OF CAJAL TO SECTIONS OF THE RETINA¹

DR. FELIX FERNANDEZ BALBUENA

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1. *Rolling up the Retina Around the Optic Disc.*—If the animals are small, we leave the retinal block resulting from the rolling up adhering by means of the optic nerve to a round segment of the eyeball. In the large retinae of the bull or sheep we make several blocks, in order to facilitate the penetration of the fixation fluid.

2. *Fixation of the retinal blocks* in absolute alcohol (50 c.c.) with pyridin (5 to 10 c.c.), according to formula 2°B of Cajal. We also use the absolute alcohol alone, and ammoniacal alcohol, as in the classic method. The retinal blocks remain in the fixation fluid twelve, twenty-four, or forty-eight hours, according to the thickness of the specimens.

3. When the specimens are once fixed, they are enclosed in celloidin and mounted *on corks*. They are then immersed in 70 per cent. alcohol and the sensibilization of the specimens is ascertained. The blocks mounted *on cork* should remain in 70 per cent. alcohol from a few days to two or three months.

The best results are usually obtained within the first month. This third operation is usually decisive. We call it provisionally *sensibilization* of the *retinal* block, since it appears that, without a more or less prolonged impregnation of the blocks in 70 per cent. alcohol, the succeeding steps of nitration, developing, and fixing of the sections will be failures.

4. *Impregnation of the Sections in a Nitrate of Silver Solution.*—After the retinal blocks have undergone the impregnation process described, sections are made which are grasped with a paint-brush and deposited on the bottom of a porcelain or glass vessel still moistened with the alcohol which we have used for lubricating the blade of the microtome; then 5 c.c. or more of a 1:2000 silver nitrate solution to which

¹ Special demonstration at the Army Medical Museum.

a few drops of pyridin (5 or 10 drops—20 c.c.) have been added, are poured over the sections.

When the sections are saturated, we slowly heat the solution until a slight steam rises, whereupon we let the solution cool off.

Sometimes it happens that the section does not assume a yellow color, as is the case when the conditions are favorable to impregnation. In such cases the solution is heated several times until the yellow color appears. Generally three to ten minutes of embedding in the nitrate of silver bath are sufficient.

5. *Preparation of the Colloid Medium and Developing of the Sections.*—Three or four drops of tincture of yellow amber added to the nitrate of silver bath impart to the solution a colloid consistence. Afterward two or three drops of a 1 per cent. hydroquinone solution are added, at the same time shaking the vessel to render the mixture homogeneous.

When the reaction is favorable, a gradual deepening of the color of the sections and a clear differentiation of the various regions are noticed. The time in which the sections remain in the developing fluid varies from four to ten minutes. By watching the effects we find when we have to terminate the process. By some calculation we easily arrive in each case at the desired results. In some cases it is necessary to assist the reaction by slow heating on the alcohol lamp.

6. *Fixation.*—When the sections are developed, they are washed in the same vessel with distilled water. Then we pour on them a 1 per cent. borax solution and three or four drops of a 1 per cent. yellow chlorid of gold solution. We finish the process by fixation in a 5 per cent. hyposulphite solution.

AN INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

MINUTES OF THE PROCEEDINGS

TUESDAY MORNING, APRIL 25, 1922

The first session was called to order in Colonial Hall of the Daughters of the American Revolution, by the Chairman of the Committee on Arrangements, Dr. W. H. Wilmer, Washington, D. C., and an address of welcome was made by Hon. Calvin D. Coolidge, Vice-President of the United States.

After roll call of delegates to the Congress the committee appointed to nominate permanent officers of the Congress made its report, which was unanimously adopted, and the officers named were declared elected (page 5).

The Committee of Arrangements made the following report:

Owing to the fire at the Hotel Willard it has been necessary to make extensive changes in the plans for the Congress. The scientific sessions and the evening addresses will be held in Colonial Hall, Daughters of the American Revolution.

On Tuesday afternoon there will be an official trip to Mount Vernon by trolley. A wreath will be placed upon the tomb of Washington.

On Tuesday evening, at the Corcoran Gallery of Art, the President and the Officers of the Congress will hold a reception in honor of the members of the Congress and ladies.

On Wednesday afternoon the President of the United States will receive the members of the Congress and ladies at the White House.

Members may also visit the Pan American Union Building, the Army Medical Museum, Library of Congress, and other places of interest.

On Thursday afternoon there will be an official visit to the Bureau of Standards, and demonstrations will be given at the Army Medical Museum.

On Friday evening a banquet will be held at the Hotel Willard.

The Report of Committee on Scientific Business was as follows:

The Report of the Committee on Scientific Business consists of the pre-session volume, which most of you have received, and the program of over one hundred pages. This was possible only through the co-operation of another committee, the Committee on Publication, who have most nobly

carried on our work. The third part of our work is the Daily Bulletin, which will be ready for distribution each morning.

The work of securing a scientific program has been arduous, but it has been a most grateful task, one which has brought us into communication with some of the great men in ophthalmology of our age, and we hope to share with all the members of the Congress an even more intimate communication in the three days that are to come.

THE PRESIDENT: We will now proceed with the scientific discussions. The first paper is entitled "*Des Enseignements de la Guerre en Chirurgie Oculaire*" (Teachings of the War in Ocular Surgery), by Prof. F. de Lapersonne, of Paris.

The paper of Prof. de Lapersonne was discussed by Drs. Frederick T. Tooke, George S. Derby, Allen Greenwood, G. F. Rochat, Edmond E. Blaauw, and Prof. F. de Lapersonne.

The paper of Sir William Lister, on "Detachment of the Vitreous," was read by Mr. E. Treacher Collins, who said: I first of all have to express to you Sir William Lister's sincere regret that he is unable to be present. The state of his health prevented his undertaking the journey. When he asked me to read an abstract of his paper I undertook to do so with a light heart because I felt sure that any deficiency which there might be on my part would be fully compensated for by the beautiful lantern slides which he had given me to show.

The paper of Sir William Lister was discussed by Drs. Marcus Feingold, Thomas B. Holloway, John E. Weeks, J. W. Nordenson, Ignacio Barraquer, F. Park Lewis, F. H. Verhoeff, Col. R. H. Elliot, Mr. E. Treacher Collins and Prof. E. Gallemaerts.

The next paper was read by Prof. Allvar Gullstrand, of Upsala, Sweden, on "*On Diaphragm Lamps in Ophthalmology.*"

The paper of Prof. Gullstrand was discussed by Drs. Edward Jackson, Harry S. Gradle, E. E. Blaauw, G. F. Rochat and Prof. Allvar Gullstrand.

The paper by Dr. José de Jesus Gonzalez, of Mexico, on "*Síntomas Oculares del Envenenamiento por el Piquete de Alacrán*" (Ocular Symptoms of Poisoning by the Sting of a Scorpion), was read by Dr. J. M. Penichet.

The paper of Dr. Gonzalez was discussed by Drs. Francisco M. Fernandez, Aurelio Beraun, Lloyd Mills, and Col. R. H. Elliot.

At the conclusion of the paper of Dr. Gonzalez the Congress adjourned.

WEDNESDAY MORNING, APRIL 26, 1922

The Congress was called to order by the President, Dr. George E. de Schweinitz.

Dr. S. Gemblath, of Paris, read a paper on "*Traitement du Trachome par les Injections sous Conjonctivales de Cyanure de Mercure*" (Treatment of Trachoma by Subconjunctival Injections of Mercury Cyanid).

The paper of Dr. Gemblath was discussed by Drs. Jesus M. Penichet, Joseph Krinsky, John McMullen, Lucien Howe, Prof. F. de Lapersonne, and Dr. Said Gemblath.

The President announced the following members on the committees to take in charge the consideration of propositions which were brought before the Congress for action, with the understanding that they should divide their energies between the three problems:

The Value of Letters and Characters as Visual Tests:

Dr. Edward Jackson, Denver, Colorado.

Dr. A. E. Ewing, St. Louis, Mo.

Dr. G. F. Rochat, Groningen, Holland.

Ophthalmic versus Anatomic Nomenclature:

Dr. M. Uribe Troncoso, New York City.

Dr. M. Feingold, New Orleans.

Dr. J. W. Nordenson, Stockholm, Sweden.

Standards for Field Taking:

Col. Robert H. Elliot, London, England.

Dr. Luther C. Peter, Philadelphia, Pa.

Dr. A. Magitot, Paris, France.

Prof. C. E. Ferree, Bryn Mawr, Pa.

THE PRESIDENT: It is the Chair's understanding that these three committees are combined in one, each section having its special duty. A report will be made which shall be regarded as a preliminary one. A more extended study will be reported at a future Congress. The Chair suggests that the Secretary of the Congress, himself the author of one of the propositions, shall be requested to communicate with the various members as to their meetings.

The Chair has the honor to ask the senior Vice-President of the Congress, Dr. Lucien Howe, to take the chair.

DR. LUCIEN HOWE: The next paper on "Anomalies of Ocular Development and Pigmentation: 1. Bilateral Symmetric Congenital Corectopia with Iridodonesis and Microcoria; Microlentis and Coloboma Lentis. 2. Melanosis of the Eye, Skin of Right Side of Head, and Right Palate," will be presented by Mr. J. Gray Clegg, of Manchester, England.

The paper of Dr. Clegg was discussed by Mr. E. Treacher Collins and Drs. E. E. Blaauw, Edward Jackson, and J. Gray Clegg.

DR. LUCIEN HOWE: The next paper, "Bitemporal Contraction of Visual Fields in Pregnancy," will be presented by Prof. C. E. Finlay, of Havana, Cuba.

The paper of Prof. Finlay was discussed by Drs. Walter B. Lancaster, Maud Carvill, and Profs. de Lapersonne and Finlay.

The President, Dr. de Schweinitz, then took the Chair, and announced that the next paper on the scientific program was "Les Échanges d'Eau et d'Ions à Travers la Cornée," by Dr. V. Morax, of Paris. Dr. Weeks stated

that Dr. Morax was unable to attend on account of illness in his family, but that he wished to convey his greetings to the members of the Congress and to express his regrets and his apologies for his inability to be here.

The next paper was by Dr. Francisco Poyales, of Madrid, entitled "La Tuberculosis Ocular Infantil" ("Infantile Ocular Tuberculosis").

The paper of Dr. Poyales was discussed by Drs. W. C. Finnoff, J. Gray Clegg, Ignacio Barraquer, H. H. Stark, John E. Weeks, E. Treacher Collins, Richard Kerry, F. H. Verhoeff, and Francisco Poyales.

Prof. F. de Lapersonne, of Paris, at the request of the President, took the Chair and announced the paper of Dr. C. F. Harford, of Cambridge, on "Psychopathology in Ophthalmic Practice," which was read by Mr. J. Gray Clegg.

The paper of Dr. Harford was discussed by Drs. George F. Keiper and J. Gray Clegg.

Dr. B. Castresana, of Madrid, was not present to read his paper entitled "Nuevo Tratamiento Quirúrgico del Estrabismo" ("Treatment of Strabismus by a New Surgical Procedure").

Dr. E. Campodonico, Lima, Peru, was not present to read his paper entitled "A New Procedure in the Excision Method of Pterygium Operation."

Dr. S. Lewis Ziegler, Philadelphia, read a paper entitled "The Subconjunctival Excision of Pterygium."

The papers of Drs. Campodonico and Ziegler were discussed by Drs. John O. McReynolds, A. E. Prince, and S. Lewis Ziegler.

WEDNESDAY AFTERNOON, APRIL 26, 1922

By the permission and courtesy of Brig.-Gen. Robert E. Noble the following demonstrations were given at the Army Medical Museum, the arrangements being in charge of Mayor G. R. Callender.

APPARATUS AND METHODS OF EXAMINATIONS

MR. E. TREACHER COLLINS, London, England: Lantern Slide Demonstration.

PROF. ALLVAR GULLSTRAND, Upsala, Sweden: Diaphragm Illumination.

DR. J. W. NORDENSON, Stockholm, Sweden: Centric Photography of the Fundus Oculi.

PROF. I. BARRAQUER, Barcelona, Spain: Moving Pictures of the Operation of Extraction of Cataract in the Capsule.

COL. ROBERT H. ELLIOT, M.D., London, England: The Elliot Sign of Glaucoma.

DR. GEORGE MACKAY, Edinburgh, Scotland: Clockwork and Cable Trephines.

DR. JOHN A. MCCAW, Denver, Col.: A Rotating Campimeter for Mapping Paracentral Scotomas.

LUTHER C. PETER, M.D., Philadelphia, Pa.: Perimetry.

- DR. J. ELLIS JENNINGS, St. Louis, Mo.: Improved Test for Color-Blindness.
 DR. W. H. CRISP, Denver, Col.: Photographic Demonstration of Crossed Cylinder Test for Astigmatism.

PATHOLOGIC SPECIMENS AND MICROSCOPIC SLIDES

- Exhibit from the Museum of the American Academy of Ophthalmology and Oto-Laryngology.
 MR. J. GRAY CLEGG, Manchester, England: Miscellaneous Specimens for Self and Colleagues.
 PROF. W. GORDON M. BYERS, Montreal, Canada: Acute Tubercular Uveo-keratitis.
 PROF. M. F. GUYER, Madison, Wis.: Experimental and Transmitted Eye Defects.
 DR. LUCIEN HOWE, Buffalo, N. Y.: Eyes of Rabbits in Which Hereditary Blindness Has Been Produced.
 PROF. ALBERTS DEL MONTE, Naples, Italy: Protozoan Bodies Discovered and Demonstrated in Chalazion.
 DR. FELIX F. BALBUENA, Gijon, Spain: A New Technic for the Application of the Method of Cajal to Sections of the Retina.
 DR. F. H. VERHOEFF, Boston, Mass.: a. Characteristic Lesions and Organisms (Leptothrices) of Parinaud's Conjunctivitis; b. Microscopic Specimen of Coralliform Cataract Showing Protein Crystals in the Lens; c. Microscopic Specimen of Asteroid Hyalitis.
 DR. E. V. L. BROWN, Chicago, Ill.: Massive Band of Connective Tissue Surrounding the Choroidea in a Traumatized Shrunk Eye.
 DR. W. C. FINNOFF, Denver, Col.: Tuberculosis as It Appears in the Eye.
 DR. CASEY A. WOOD and LIEUT.-COL. F. H. GARRISON: An exhibit of books of historic interest to ophthalmologists arranged in the Library of the Surgeon-General's Office.
 DR. A. MAGITOT demonstrated in the auditorium of the Corcoran Art Gallery a series of Lantern Slides Showing Development of the Human Eye.

WEDNESDAY EVENING, APRIL 26, 1922

The Congress met at the Colonial Hall, and the following addresses were delivered:

- PROF. S. E. WHITNALL, McGill University, Montreal, "Some Descriptive Errors in the Anatomy of the Orbit."
 PROF. J. PARSONS SCHAEFFER, Jefferson Medical College, Philadelphia, "On the Clinical Anatomy of the Efferent Lacrimal Passageways."

THURSDAY MORNING, APRIL 27, 1922

The Congress was called to order by the President, Dr. George E. de Schweinitz.

The Chair announced that the Chairman of the Section on Oph-

thalmology of the American Medical Association wished to extend to the foreign guests an invitation to attend that meeting in St. Louis, May 24 to 26, inclusive. A similar invitation was extended by the President of the American Ophthalmological Society to attend the meeting of the Society in Washington, D. C., May 2-4 inclusive.

Drs. F. H. Verhoeff and A. N. Lemoine, of Boston, read a paper on "Endophthalmitis Phacoanaphylactica."

The paper of Drs. Verhoeff and Lemoine was discussed by Drs. Lucien Howe, G. F. Rochat, James M. Patton, L. C. Rood, Arnold Knapp, George S. Derby, Albert N. Lemoine, and F. H. Verhoeff.

Prof. Emile Gallemaerts, of Brussels, Belgium, at the request of the President, took the Chair.

PROF. GALLEMAERTS: The next paper is by Dr. A. E. Davis, of New York City, on "Serum and Vaccine Treatment for the Prevention and Cure of Cataract."

The paper of Dr. Davis was discussed by Drs. Lucien Howe, M. F. Guyer, Henry H. Tyson, F. A. Davis, and A. E. Davis.

PROF. G. F. ROCHAT, Gronigen, Holland, at the request of the President, took the Chair, and announced that the papers of Prof. Barraquer and Prof. Gallemaerts would be discussed together. Prof. Barraquer's paper is entitled "Facoerisis" ("Phacoerisis"), and Prof. Gallemaerts' paper is on "Operation de Barraquer" ("Operation of Barraquer").

The papers of Professors Barraquer and Gallemaerts were discussed by Drs. Lucien Howe, John Westley Wright, John O. McReynolds, L. D. Green, Col. R. H. Elliot, Prof. F. de Lapersonne, Drs. Lloyd Mills, Joseph A. White, J. Gray Clegg, S. Gemblath, Francisco Poyales, W. A. Fisher, and Professors Barraquer and Gallemaerts.

PROF. G. F. ROCHAT: The paper of Dr. D. Kostitch, Belgrade, Serbia, entitled "Troubles Oculaires Consecutifs à l'Observation Directe de l'Éclipse Solaire" ("Ocular Troubles Following Direct Observation of the Solar Eclipse"), will be read by the Secretary.

The paper of Dr. D. Kostitch was discussed by Col. R. H. Elliot, Drs. George MacKay, J. Gray Clegg, and Francisco M. Fernandez.

PROF. G. F. ROCHAT: The next paper on "Orbital Marsupialization and Superiority of Organic Grafts of Dead Tissue in Establishing a Mobile Stump," will be read by Dr. A. Magitot, of Paris.

The paper of Dr. Magitot was discussed by Drs. J. N. Roy and A. Magitot.

DR. EDWARD JACKSON, Denver, Col., at the request of the President, took the Chair.

DR. EDWARD JACKSON announced the next paper by Dr. A. F. Alonso, of Mexico, entitled "La Peritomia en las Ulceras Crónicas Vasculares de la Cornea" ("Chronic Vascular Ulcers of the Cornea").

The paper of Dr. Alonso was discussed by Drs. L. Webster Fox, S. Lewis Ziegler, and A. F. Alonso.

DR. EDWARD JACKSON: The last paper on the program is by Dr. R. C. Cheney, of Boston, on "Types of Pneumococcus Found in Corneal Ulcers."

The paper of Dr. Cheney was discussed by Drs. George S. Derby and R. C. Cheney.

THURSDAY EVENING, APRIL 27, 1922

The Congress met in Colonial Hall, and the following addresses were delivered:

PROF. LEONARD T. TROLAND (Harvard University): "The Facts and Theories of Color Vision."

PROF. M. G. GUYER (University of Wisconsin): "The Production and Transmission of Certain Eye Defects."

FRIDAY MORNING, APRIL 28, 1922

The session was called to order by the President, Dr. George E. de Schweinitz.

COL. R. H. ELLIOT, London, England, read a paper on "The Diagnosis of Glaucoma."

The paper of Col. Elliot was discussed by Drs. John E. Weeks, Luther C. Peter, Mr. E. Treacher Collins, Drs. E. E. Blaauw, J. W. Nordenson, J. Gray Clegg, George F. Keiper, Arnold Knapp, Col. R. H. Elliot, and Dr. George W. Jean.

Prof. C. Charlin, Santiago, Chile, was not present to read his paper, "L'État Vasculaire des Glaucomateux" ("Étude de 100 Malades de Glaucome Primitif") ("Vascular Condition of Glaucomatous Patients").

MR. E. TREACHER COLLINS took the Chair at the request of the President, and announced that the paper, "Blood-Pressure in the Vessels of the Eye," by Drs. A. Magitot and P. Bailliart, of Paris, would be read by Dr. Magitot.

MR. E. TREACHER COLLINS: The next paper is by Dr. J. M. Wheeler, of New York City, on "Plastic Operations About the Eye."

The paper of Dr. Wheeler was discussed by Drs. W. E. Lambert, John E. Weeks, and John M. Wheeler.

MR. E. TREACHER COLLINS: Prof. J. N. Roy, of Montreal, Canada, will read his paper on "Lagophtalmie Bilatérale Consécutive à la Perte Accidentelle de la Peau du Front, et du Cuir Chevelu. Blépharoplastie" ("Bilateral Lagophthalmos Following Accidental Loss of the Scalp. Blepharoplasty").

THE PRESIDENT, DR. DE SCHWEINITZ, in the Chair. The next paper, by Mr. A. S. Percival, of Newcastle-on-Tyne, entitled "Light Sense: The Practical

Significance of Its Variations: Simple Tests for Determining Them," will be read by Dr. Alexander Duane, of New York City, in the absence of Mr. Percival.

The paper of Mr. Percival was discussed by Drs. C. E. Ferree, Otto Roelofs, Col. R. H. Elliot, and Dr. Alexander Duane.

PROF. C. E. FINLAY, Havana, Cuba, took the Chair at the request of the President, and announced the next paper by Drs. C. E. Ferree and G. Rand, of Bryn Mawr, Pa., "Some Contributions to the Science and Practice of Ophthalmology."

The paper of Drs. Ferree and Rand was discussed by Drs. Alexander Duane, Luther C. Peter, William L. Benedict, William Zentmayer, J. Gray Clegg, and C. E. Ferree.

PROF. C. E. FINLAY: As Dr. E. Junès, of Sfax, Tunis, is not present, the next paper is on "Carcinoma of the Choroid," by Dr. Allen Greenwood, of Boston. The paper of Dr. A. J. Bedell, of Albany, N. Y., on "Chloroma," will follow, and these papers will be discussed together.

The papers of Drs. Greenwood and Bedell were discussed by Dr. George E. de Schweinitz.

FRIDAY AFTERNOON, APRIL 28, 1922

BUSINESS SESSION

The meeting was held at the building of the Medical Society of the District of Columbia.

THE PRESIDENT: The first order of business is a resolution which Dr. Lucien Howe wishes to present:

WHEREAS, Arrangements for any International Congress necessitate much time and careful attention; and,

WHEREAS, The destruction by fire of the hall and part of the hotel agreed upon for the meeting involved many and important changes, just as the Congress began; and,

WHEREAS, That emergency was met so promptly and efficiently by the local committee as not to disturb our delightful sojourn in Washington; therefore be it

Resolved, That we express our hearty thanks to the chairman and members of the Committee on Arrangements for making these meetings, from first to last, so eminently satisfactory, and,

Resolved, That our thanks be extended also to each of the chairmen and members of the various committees whose united efforts have proved so efficient in causing this to be long remembered as the largest and one of the best organized of any International Congress of Ophthalmology.

MR. E. TREACHER COLLINS (London, England): On behalf of my colleagues, the British representatives to this meeting, I wish to express our most emphatic appreciation of this resolution and to cordially support it. We are very grateful to the officers for the care and attention which they have given

to the carrying out of this meeting, and we wish to express our special indebtedness to them.

The motion was unanimously carried.

The Secretary was directed to spread the resolution upon the minutes of the Congress, and to send a copy to the chairman of each special committee.

MR. E. TREACHER COLLINS: I wish to read a letter from the President of the Ophthalmological Society of the United Kingdom, Mr. J. Herbert Fisher.

11 Chandos Street
Cavendish Square, W. 1.
17th March, 1922

Dear Mr. Collins:

International Congress of Ophthalmology.

The Council of this Society strongly approves of the proposed suggestion to hold the next International Congress in London in 1925.

It has been in communication with the following ophthalmic bodies, which have all given their approval and promised their assistance toward ensuring success to such a meeting, namely:

Ophthalmic Section, Royal Society of Medicine.

Oxford Ophthalmological Congress.

Midland Ophthalmological Society.

North of England Ophthalmological Society.

Irish Ophthalmological Society.

Scottish Ophthalmological Club.

Such a consensus of opinion may be taken to represent the views of British ophthalmic surgeons, and the Council of this Society has instructed me to ask you to convey an invitation (enclosed herewith) to the International Congress of Ophthalmology at Washington.

A similar congress has not been held in Great Britain for many years, and, if the proposal is agreed to, it would be esteemed a high honour to British ophthalmology.

Yours sincerely,
(Signed) J. HERBERT FISHER,
President.

I will hand to the President a letter addressed to him.

THE PRESIDENT: This letter reads as follows:

11 Chandos Street,
Cavendish Square, W. 1.
17th March, 1922

Dr. G. E. de Schweinitz,
Chairman of the General Committee,
International Congress of Ophthalmology,
Washington.

Dear Sir:

Ophthalmologists in this country are very desirous that the next International Congress should be held in Great Britain in 1925; and we shall esteem it a high honour if the Congress at Washington will accept this invitation, which our colleague, Mr. E. Treacher Collins, has been asked to convey.

This proposal has met with unanimous approval not only by the Council of this Society, but also by the Councils of the following:

- (1) Ophthalmic Section, Royal Society of Medicine, London.
- (2) Oxford Ophthalmological Congress.
- (3) Midland Ophthalmological Society.
- (4) North of England Ophthalmological Society.
- (5) Irish Ophthalmological Society.
- (6) Scottish Ophthalmological Club.

We beg to remain, dear Sir,

Yours obediently,

(Signed) J. HERBERT FISHER,
President.

J. F. CUNNINGHAM,
FRANK JULER,
Secretaries.

PROF. EMILE GALLEMAERTS (Brussels, Belgium): The Ophthalmological Society of Belgium asked me to bring before you the same invitation as that of Mr. E. Treacher Collins, but in the face of so great an invitation from Mr. Collins I can only present the hope that in the course of time such invitation will be received with the applause accorded the invitation of Mr. Collins.

THE PRESIDENT: The Chair feels sure the time will never come when an invitation from Belgium will not be warmly applauded. The Chair permits himself to suggest that, before proceeding with this matter, someone shall move a vote of thanks to Professor Gallemaerts, which he in turn may convey to his colleagues in Belgium.

The motion was made by Prof. Allvar Gullstrand, seconded, and unanimously carried.

PROF. ALLVAR GULLSTRAND (Upsala, Sweden): We have received an invitation from our colleagues of England, and I should like to point out a few hints at this opportunity. I am the official delegate of the Government of a country that was strictly neutral during the War, and I can prove that I was strictly neutral, for I have received, during the War, honorary memberships from both sides—one from Great Britain and one from Austria, and that would not have happened if I had not been neutral. Now we neutrals stand at a corner—on the one side we see Germany, who thinks you are boycotting her science and her language; and on the other side we see yourselves, and we know *you are not*.

I move in the first place that this Congress shall accept the kind invitation that we have received from the Ophthalmological Society of the United Kingdom; and in the second place that this Congress shall express its wish that the next Congress is to be strictly international, and that the German language shall be an official language among others.

DR. LUCIEN HOWE (Buffalo, N. Y.): In rising to second this motion made by Professor Gullstrand I desire to do so in words which will tend to clear up an evident misunderstanding concerning the languages to be used at this Congress. We all know that when the first steps were taken toward its organization, over two years ago, an invitation was transmitted through the

Department of State to each of the countries with which we were then in diplomatic relations, requesting that a representative be assigned to the Congress, and therefore such an invitation could not go to those countries with which such diplomatic relations did not exist at that time.

This probably led to the impression, unfortunately widespread and often repeated, that we Americans had "boycotted" the German language. The fallacy of this, however, was shown by the fact that in one of the earliest circulars issued by our Central Committee it was distinctly stated that any language could be used which the speaker preferred, provided it be translated into one of the three official languages of the Congress.

Also, it seemed probable then that very few colleagues would come just now from Germany or Austria, and the printing of notices and of transactions in four languages would involve a deficit even greater than the one of several thousand dollars which we are now obliged to supply for the publication in only three languages.

Therefore it seems proper for some one to prove that this is an International Congress, not only in name, but that it is catholic in its scope and hospitable to ideas, no matter whence they come. In other words, the German language should purposely be used by some one who was born and bred in America and without a trace of German predilection. We need that as a proof of consistency. Speaking in German, Dr. Howe continued:

Deshalb, Herr Präsident, möchte ich meiner Meinung dahin Ausdruck geben, dass solange wir gewillt sind die Arbeiten der Förscher anzuerkennen, und unsere Kenntnisse dadurch zu bereichern, so müssten wir doch deutlich zeigen dass wir nicht ihre Sprache verbannen. Es ist zwar möglich, dass diese Konsequenz, auf die ich hinweise, von den Anwesenden missverstanden werden, ebenso wie wir seinerzeit von den Deutschen misverstanden wurden, ich bin aber bereit irgend welchen Beschuldigungen entgegen zu treten. Ich behaare also auf das Recht, wenn ich es wünsche, die deutsche, oder irgend eine andere fremde Sprache zu gebrauchen.

Indem ich meine Zustimmung für die Annahme der Einladung nach London abgebe, hoffe ich, gemeinsam mit Prof. Gullstrand und will es auch ev. für die meisten der englischen Kollegen hoffen, dass beim nächsten internationalen Ophthalmologen Kongress Deutsch als eine der offiziellen Sprachen gelten wird.

DR. GEORGE MACKAY (Edinburgh, Scotland): I rise to support the proposition put before you by Mr. E. Treacher Collins, speaking in behalf of all the ophthalmologic colleagues in Great Britain, and I trust I may say of Ireland, inviting you most cordially to accept this invitation to make your next meeting place the capital of the British Empire. The last name in the list read is that of the Scottish Ophthalmological Club. It is a small body, but I venture to say it is a very select body. It represents a country which has ever stood for liberty, for progress, for freedom. I take it, gentlemen, that that is a sentiment, an aspiration, which appeals to you all. I beg, then, to assure you that in visiting the British Isles you come not only to receive a hearty welcome from London and from England, but from bonnie Scotland, to which you will find time to extend your visit.

DR. JOHN ROWAN (Glasgow, Scotland): I wish to second Mr. E. Treacher Collins' invitation, which I hope the Congress will accept, and which I wish to back up from the point of view of the Scotch colleagues. We hope that while you are there, either going or coming, you will take the northern route. There are plenty of trains, three or four lines, and it is only about an eight-hour trip. We assure you the clinics will be open to you and we will do our best to show you all our different methods. I am afraid, however, by the time you have attended the meetings in London the wives and daughters will have taken command and that you will be taken to the north of Scotland for sight-seeing. We will do our best in that way, too. If you will let us know what you want, we will be pleased to do it. We will try to live up to the standards that have been set at this Congress, although that will be difficult. The work has been extremely good, the organization has been good, and the discipline has been good. There has been no time wasted.

MR. J. GRAY CLEGG (Manchester, England): There is no delicate question in the north of England as regards nationality, and as representing my colleagues of the North of England Ophthalmological Society I wish to add our strong invitation and to express the hope that this Congress will accept it. The stimulus of this visit to America will certainly set the north of England ball rolling at a great pace, and I expect we will be able to have something to show you in spite of the fact that perhaps we have not been quite up to the mark in times gone by. I have pleasure, therefore, in expressing the hope of the North of England Ophthalmological Society that you will accept the invitation.

COL. R. H. ELLIOT (London, England): Nothing has been said about Wales and very little about Ireland, but I am both Welsh and Irish and I am sure both will be as glad to see you come as any of us.

THE PRESIDENT: The invitation conveyed by Mr. E. Treacher Collins, which bears the endorsement of the various societies enumerated, is now before you. Does the Congress understand that it includes the wish expressed in Professor Gullstrand's motion?

An affirmative answer having been recorded, the Congress by vote accepted the invitation to hold the next meeting in England in 1925.

THE PRESIDENT: Mr. Collins, will you please convey to your confrères and the societies enumerated the acceptance by this Congress of Great Britain's invitation? Is there any other business before the Congress?

DR. C. D. WESCOTT (Chicago): As a member of the Hospitality Committee of Chicago I wish to extend an invitation to all our guests and to any members of the Congress to come to Chicago. We will be glad to do anything we can to assist you in seeing anything we have to show you.

SCIENTIFIC SESSION

THE PRESIDENT: The first number in our scientific program this afternoon is a paper on "Metastatic Thyroid Tumor in the Orbit," by Dr. Arnold Knapp, of New York City.

The next paper was by Mr. E. Treacher Collins, of London, "Hereditary Ocular Degenerations—'Ophthalmic Abiotrophies.'"

The paper of Mr. Collins was discussed by Drs. C. E. Finlay, F. H. Verhoeff, George F. Libby, Mary Buchanan, William E. Bruner, Mr. E. Treacher Collins, Edward Jackson, and E. E. Blaauw.

Drs. Walter Scott Franklin and Frederick C. Cordes presented a paper on "Bilateral Lymphosarcoma of the Orbit with Intermittent Exophthalmos."

DR. A. TORRES ESTRADA, of Mexico City, was not present to read his paper, "Mercurials and Salvarsan and its Derivatives in the Treatment of Grave Eczematous Conjunctivitis and Keratitis."

The next paper, "The Effects of Direct Instillation of Novarsenobillon in the Conjunctival Sac in Resistant Cases of Congenital Syphilitic Interstitial Keratitis," by Dr. A. Renshaw, of Manchester, England, was read by Mr. J. Gray Clegg.

DR. EDWARD JACKSON made the following report:

The Committee appointed to report on Suggestions for Congress Action concluded it was quite impossible to give either of the three questions referred to it any adequate consideration during the time of this Congress, and recommend that it be authorized to continue its consideration and to report either to a subsequent Congress or through the journals. After the action taken today probably the report should be made when the Congress meets in London. That is the only report the committee has to make.

The final paper on the scientific program was "Treatment of Infectious Keratitis by Vaccine," by Dr. Rovirosa Virgili, of Madrid, Spain. Dr. Virgili was not present to read the paper.

DR. ALLEN GREENWOOD (Boston): I wish to move a rising vote of thanks to the President for the very able manner in which he has conducted the sessions of this Congress. And I would like to give the same vote of thanks to our Secretary.

The motion seconded by Mr. J. Gray Clegg, and unanimously carried.

THE PRESIDENT: Ladies and Gentlemen of the Congress: This concludes the scientific program. I beg on behalf of my colleagues to express our best thanks for your courtesy and your kindness and to reiterate our high appreciation of your sympathetic interest and your notable scientific contributions which have brought success to the Congress of Ophthalmology.

ADJOURNMENT.

ORGANIZATION OF THE CONGRESS

In 1919, the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-Laryngology each appointed a committee of three members, with instructions to formulate plans for the organization of an International Congress of Ophthalmology to meet in the United States. The membership of these committees was as follows:

American Ophthalmological Society, Drs. G. E. de Schweinitz, W. H. Wilmer, and Frederick T. Tooke.

Section on Ophthalmology of the American Medical Association, Drs. W. H. Wilder, Lucien Howe, and Francisco Fernandez.

American Academy of Ophthalmology and Oto-Laryngology, Drs. Edward Jackson, W. B. Lancaster, and Luther C. Peter.

At a meeting in New York, on October 21, 1919, an organization of the combined committees was effected with the election of the following: Dr. G. E. de Schweinitz, Chairman; Dr. Edward Jackson, Vice-Chairman, and Dr. Luther C. Peter, Secretary. A temporary Sub-committee on Time and Place was appointed, which at a subsequent meeting recommended that the place of meeting should be Washington, D. C., April 18, 1922.* This recommendation was adopted at a later meeting of the general committees of the Congress. Numerous other sub-committees were appointed by the Chairman, selected to represent all sections of the United States, as well as Canada, Cuba, Mexico, and Central and South America.

The several committees in charge of the organization of the Congress, as finally appointed, consisted of a General Committee, a Committee on Organization, a Committee on Scientific Business, a Committee on Arrangements, a Committee on Membership and Credentials, a Committee on Finance, a Committee on Publication, and a Committee on Entertainment for each of several leading cities throughout the country.

* The final date selected was April 25-28, 1922.

INTERNATIONAL CONGRESS COMMITTEES**GENERAL COMMITTEE**

DR. GEORGE E. DE SCHWEINITZ, CHAIRMAN

DR. EDWARD JACKSON, VICE-CHAIRMAN

DR. LUTHER C. PETER, SECRETARY-TREASURER

DR. FRANCISCO M. FERNANDEZ

DR. FREDERICK T. TOOKE

DR. LUCIEN HOWE

DR. WILLIAM H. WILDER

DR. WALTER B. LANCASTER

DR. WILLIAM H. WILMER

SPECIAL COMMITTEES**COMMITTEE ON ORGANIZATION**

DR. EDWARD C. ELLETT, CHAIRMAN

DR. HENRY D. BRUNS

PROF. R. PACHECO LUNA

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The report of the Committees on Organization as presented to each of the three component societies, was as follows:

It has been resolved, if the report of the Committees is accepted, to issue invitations as speedily as possible to join this Congress to physicians (ophthalmologists and others interested in ophthalmology) in good standing in the American Medical Association or other recognized scientific and medical societies of the United States and Canada, and to physicians (ophthalmologists) in good standing in Cuba, Mexico, Central and South America, and also to physicians through their constituted ophthalmologic societies in those foreign countries with whom we are (March 6, 1920) in diplomatic relationship.

It was further resolved that the official languages of the Congress shall be English, French, and Spanish, and that the membership fee shall be ten (10) dollars in United States money.

The report of the Committee was received and accepted by each of the three societies, and the following invitation was transmitted by the Department of State through the various embassies and legations abroad:

The American Ophthalmological Society, The Section on Ophthalmology of The American Medical Association, and The American Academy of Ophthalmology and Oto-Laryngology have the honor to invite, through his Excellency, the Minister of Foreign Affairs, the Government of —, to participate in An International Congress of Ophthalmology, by sending an official delegate.

The Congress will be held under the auspices of these Societies in the City of Washington, United States of America, April 25th to April 28th, 1922.

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June the first, nineteen hundred and twenty-one

This invitation was sent to the following countries:

Argentina, Belgium, Bolivia, Brazil, Bulgaria, Chile, China, Colombia, Costa Rica, Cuba, Czecho-Slovakia, Denmark, Ecuador, Finland, France, England, Egypt, Greece, Guatamala, Honduras, Italy, Japan, Morocco, Netherlands, Nicaragua, Norway, Panama, Paraguay, Persia, Peru, Poland, Portugal, Roumania, Salvador, Kingdom of Serbs, Croats and Slovenes, Siam, Spain, Sweden, Switzerland, Turkey, Uruguay, Venezuela.

The Committee on Membership sent invitations to the leading members of the profession interested in ophthalmology and to the various ophthalmological societies in these countries, and in Canada, Mexico, and the United States, asking that they make application for membership in the Congress. The members of the Congress and the societies represented are printed in the volume.

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